Journal
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

AVMA Convention—Philadelphia, August 18-21, 1958

General Articles

Sequelae of Leptospirosis in Horses on a Small Farm—*S. J. Roberts* 189
Laboratory Report on Leptospirosis in Georgia—1957— *Maurice W. Hale* 196
Comparison of Epizootic Diarrhea of Suckling Rats and a Similar Condition in Mice—*Henry L. Foster* 198
Purulent Keratoconjunctivitis in Laboratory Rats, Caused by Micrococcus Pyogenes var. Aureus—*Henry L. Foster* 201
Use of Dental Films in Veterinary Practice—*Blake—Micuda—Hubbard* ... 201
Seasonal Fluctuations of Gastrointestinal Nematodes of Imported Beef Feeder Calves in Northeast Iowa—*L. H. Petri* 203

Surgery and Obstetrics

Clinical Views on Cesarotomy in Cattle—*Jurgen Lensch* 205
Cesarotomy in the Mare—A Case Report—*Edward C. Melby, Jr.* 207
A Mandibular Splint for a Dog—*William P. Barchfeld* 209
An Ovine Oddity—*J. H. Rosenberger* 212
Magnesium Sulfate for Euthanasia in Dogs—*Jose B. Aranez and Luis B. Caday* 213

Clinical Data

The Prophylactic Effect of Chlortetracycline, Given Orally, on Bovine Leptospirosis—*Leif M. Ringen and William Okazaki* 214
Study of the Viability of Leptospira Pomona in Frozen Extended Bovine Semen—*R. K. Jones* 216
Lamb Losses Associated with Clostridium Perfringens Type A—*McGowan—Moulton—Rood* 219
Lipodystrophy in a Black Leopard—*A. C. Herzberger* 221
Erysipelas in a Flock of Chickens—*C. L. Vickers and Bert W. Bierer* 223
Effectiveness and Toxicity of Sustained Action Quinacrine Hydrochloride in Dogs—*Frederick Sperling* 225
What Is Your Diagnosis? ..... adv. p. 39

Nutrition

The Effect of Pelletizing on Vitamins A and E—*Bert W. Bierer and C. L. Vickers* 228

Editorial

Frustrating Terminology: Panleukopenia or What? 230

The News ..... 233 **Organization Section ..... adv. p. 24**

Washington News, adv. p. 16; Coming Meetings, adv. p. 32
Correspondence ..... adv. p. 4

Contents continued on adv. pages 2 and 4

Vol. 133 **August 15, 1958** **No. 4**

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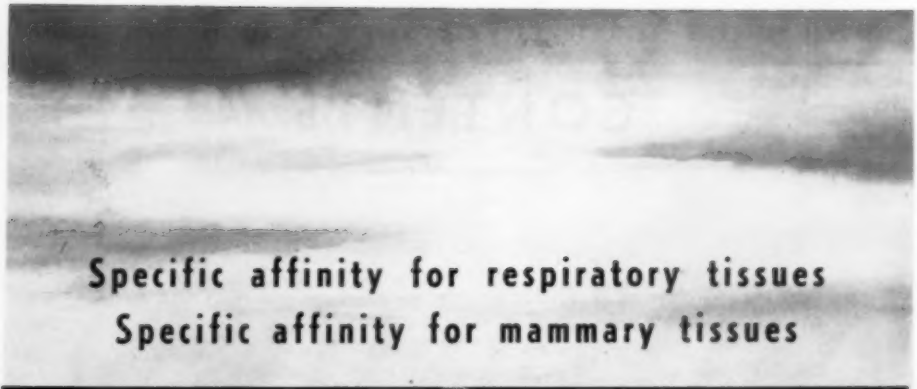

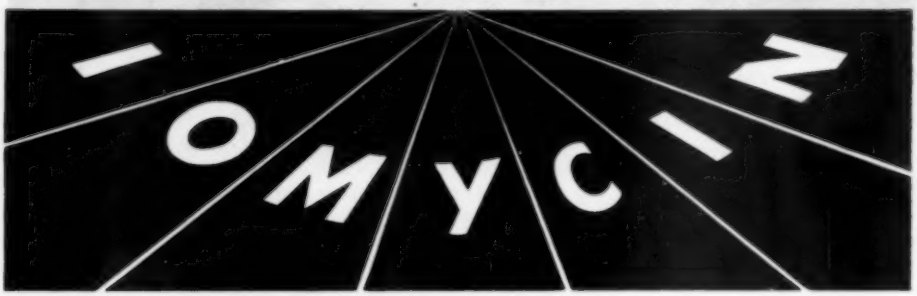

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CONTENTS

Continued from Cover

SURGERY AND OBSTETRICS

Cesarotomy in Two Mares 208
Antibody Absorption in Newborn Herbivores 209
Torsion of the Uterus in the Mare 212
Evaluation of Semen Quality in the Dog ... 213

CLINICAL DATA

Leptospiral Nephrosis in Calves 215
Porcine Leptospirosis in Romania 218
Experimental Ovine Leptospirosis 218
How Zinc May Be Lost 218
Transmission of Favus of Mice to Man 218
Spontaneous Chylopneumothorax in Man .. 222
An Intradermal Test for Turkey Ornithosis . 222
Fluoridated Water and Chick Growth 224
Pesticide Poisoning of Livestock 224
Organophosphate Pesticide Destroyed in the Rumen 224
Toxicity of Antibiotics in Veterinary Medicine 224
A Hemorrhagic Disease of Dogs 227
Complement-Fixation Test for Rabies 227
Control of Rabies Along the Mexican Border 227
Toxoplasmosis in Cats 227
Carbohydrates for Young Pigs 227

NUTRITION

Oils, Penicillin, and Bloating 228
Effect of Ingested Paraffins on Ruminants .. 229
Effect of Gibberellic Acid in Broiler Rations . 229
Effect of Milk Components on Calf Diarrhea 229
Toxicity of Nitrates and Nitrites for Dairy Cattle 229
Protein Malnutrition in the Pig 229

CURRENT LITERATURE

Abstracts

Development of the Heart in Bovine Embryos 231
Assessment of Bovine Liver Function 231
Elimination of Phenothiazine by Dairy Cows 231
Pulmonary Adenomatosis in Iowa Cattle ... 231
Effect of Milk on Trichostrongylid Nematodes 231
Fungi in the Crop of the Turkey 231
Pendulous Crop in the Turkey 231

Books and Reports

The Conquest of Bovine Tuberculosis in the United States 232
The Guinea Pig in Research 232

EDITORIAL STAFF: W. A. Aitken, *Editor in Chief;* Donald A. Price, *Associate Editor;* Wayne H. Riser, *Editor, Small Animal Medicine;* J. G. Hardenbergh, *Managing Editor;* Helen S. Bayless, *Assistant Editor and Advertising Manager;* Eva G. Bailey, *Assistant to the Editor;* Dinah Polen, *Assistant Advertising Manager.*

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Correspondence

June 2, 1958

Dear Dr. Aitken:

Dr. Curtis C. Starkebaum, in his article "Bawling Disease in Calves" (*J.A.V.M.A., 132,* 1958: 495) implies that death of calves within six hours after birth was caused by a patent foramen ovale. It is well known that the valve of the foramen ovale is normally not sealed in the calf until about 3 weeks of age. (C. W. Ottoway: The Anatomical Closure of the Foramen Ovale in the Equine and Bovine Heart. *Vet. J., 100,* (1944): 111, 130). The valve is closed functionally as soon as blood begins to flow through the lungs and normal blood pressure develops in the left atrium. This compresses the tubular valve against the interatrial septum. (The septum is not between the auricles as stated in the article.) There is no evidence in the article that the competence of the valve was actually tested with hydraulic pressure.

In one study, it was possible to probe a patent foramen ovale in 16 per cent of clinical normal adult cattle (G. Stroh: Untersuchen an Rinderherzen über das Offenbleiben des Foramen Ovale. *Munch. tierarztl. Wchnschr. 74,* (1923): 293-297).

s/ROBERT E. HABEL,
Ithaca, N. Y.

• • •

July 5, 1958

Dear Dr. Aitken:

Thank you for your correspondence concerning my article "Bawling Disease in Calves," and the comments by Dr. Habel.

The membrane that covers the foramen ovale is indeed interatrial. Perhaps the article should have been written to convey the concept that it is an opening allowing the mixture of blood from the right and left sides of the heart (mixing of oxygenated and nonoxygenated blood) rather than an opening between the auricles.

An interesting letter has come to me from L. W. Mahaffey, of the Animal Health Trust, Equine Research Station, Newmarket, Suffolk, England. They are engaged in research with a similar condition called "barkers" in newborn foals in which the foramen ovale was patent.

Yours truly,
s/CURTIS C. STARKEBAUM
Gunnison, Colo.

[For an abstract of Dr. Mahaffey's article on "barking" foals (*Vet. Rec.,* Dec. 7, 1957, Part 2: 1277) see the April 15, 1958, JOURNAL, p. 322.

Because the deaths occurred shortly after birth, the patent foramen ovale was not considered significant and was not mentioned in the abstract.—ED.]

CONTENTS—Continued

THE NEWS

Colonel Trum, V.C. (Ret.), U.S. Army, Appointed to Harvard Medical School Post 233
Colonel John Rust to Head Life Science Exhibit in Geneva, Switzerland 233
Dr. Willard Wright Retires—to Continue as Consultant 234
Eleventh World Health Assembly Achievements 234
Animal Care Meeting 234
Consultant Group to Poultry and Egg Industries Formed 234
State Board Examinations 234
Among the States and Provinces 235
Commencements 238
Deaths 247

MISCELLANEOUS

Leptospirosis and Periodic Ophthalmia in Horses 194
Leptospirosis in Horses in Romania .. 195
Leptospirosis in Horses 195
Leptospiras in Udders of Cows 195
Tuberculosis in Animals 195
Research with Viruses 197
Modified Viral Vaccine for Equine Respiratory Infection 197
Cross Immunity Between Canine Distemper and Rinderpest 202
Prehistoric Dog Found in Germany ..... adv. p. 43

ORGANIZATION SECTION

Dr. Scheidy Re-Elected in District II, Dr. Woelffer New Member from District III ..... adv. p. 24
Applications ..... adv. p. 26

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1. Harris, J.R., and Clarkson, T.B., Prevention of Relapses in Milk Fever, Vet. Medicine, *12*:696 (Dec. 1955)

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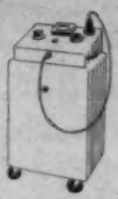

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*Chambers, E. E.: N. Am. Vet. 37:105 (Feb.) 1956.

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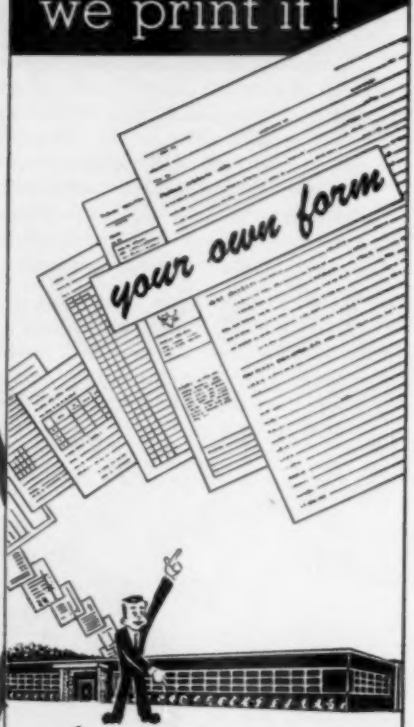

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Washington News

Legislative.—The following bills were cleared for the approval of the President: S. 3076, on the transportation of foot-and-mouth disease virus; S. 3478, referrring to the inventory date of anti-hog cholera serum (see JOURNAL, June 15, 1958, adv. p. 14); S. 2447, which directs that the Secretary of Interior undertake continuing studies on the effects of insecticides, fungicides, pesticides, etc., upon fish and wildlife; H.R. 12541, the Department of Defense Reorganization Act of 1958.

★ ★ ★ ★

New Laws:—H.R. 11414, authorizing grants to Public Health Schools for the purpose of training and service in the field of public health (P.L. 85-544); S. 385, authorizing training of federal employees at public or private facilities (P.L. 85-507); and H.R. 7963, which will amend and extend Small Business Act (P.L. 85-536).

The House-passed Supplemental Appropriations Bill (H.R. 13450, fiscal year ending June 30, 1959) included $1,750,000 for meat inspection. Should the Senate concur, the total amount available would be $19,076,000 (see JOURNAL, July 15, 1958, adv. p. 12).

The Senate, July 23, passed with amendments the House-passed bill (H.R. 7576) to further amend Civil Defense Act 1950, which would help state and local civil defense organizations meet their expenses.

The House Ways and Means Committee favorably reported bill H.R. 10, with amendment, to permit self-employed to defer from income tax, money placed in retirement annuities (see JOURNAL, Feb. 15, 1958, adv. p. 10). Bill reported would limit deductions to 10 per cent of net earnings from self-employment, but not over $2,500 in one taxable year or $50,000 in lifetime. Higher deductions would be allowed persons over 50 years of age on Jan. 1, 1959; no deduction allowed when taxpayer is over 70. Clearing the first hurdle essential for House action (which is likely if the rule is granted), there is no assurance of Senate action in the time remaining prior to adjournment of this Congress.

A Senate Subcommittee on Agriculture, Senator Humphrey (D., Minn.), chairman, held hearings on H.R. 12126 on July 22-23 (see JOURNAL, July 15, 1958, adv. p. 12). Testimony in favor of bill by U.S.D.A., U.S. Livestock Sanitary Association, National Association of State Departments of Agriculture, American Farm Bureau Federation, American National Cattlemen's Association, and the AVMA. Dr. Leonard H. Goss, president, American Association of Zoological Parks and Aquariums, and representatives from several zoological parks, public and private, requested an amendment that would permit importation of wild ruminants and swine under safeguards approved by the Secretary of Agriculture.

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New Bills:—S. Con. Res. 99, Senator Humphrey, relative to the designation of an International Health and Medical Research Year. S. Con. Res. 100, Senator Johnson (D., Texas), requesting that the President proclaim the week of Dec. 1-6, 1958, as National Science Week.

★ ★ ★ ★

Miscellaneous.—Confirmed by the Senate, July 2, 1958, to be members of National Science Board, National Science Foundation: Detlev W. Bronk, Pa.; T. Keith Glennan, Ohio; Robert F. Loeb, N.Y.; Lee A. DuBridge, Calif.; Kevin McCann, Ohio; Jane A. Russell, Ga.; Paul B. Sears, Conn.; Ernest H. Volwiler, Ill.; and Morrough P. O'Brien, Calif.

Nominated by the President to be Under-Secretary of the Department of Health, Education and Welfare: Miss Bertha S. Adkins, Maryland.

Members of the veterinary science group departed for the Soviet Union, July 22 (see JOURNAL, July 15, 1958, adv. p. 12).

New Jersey, Michigan, and New Mexico have been added to the list of states declared modified-certified brucellosis-free by U.S.D.A.

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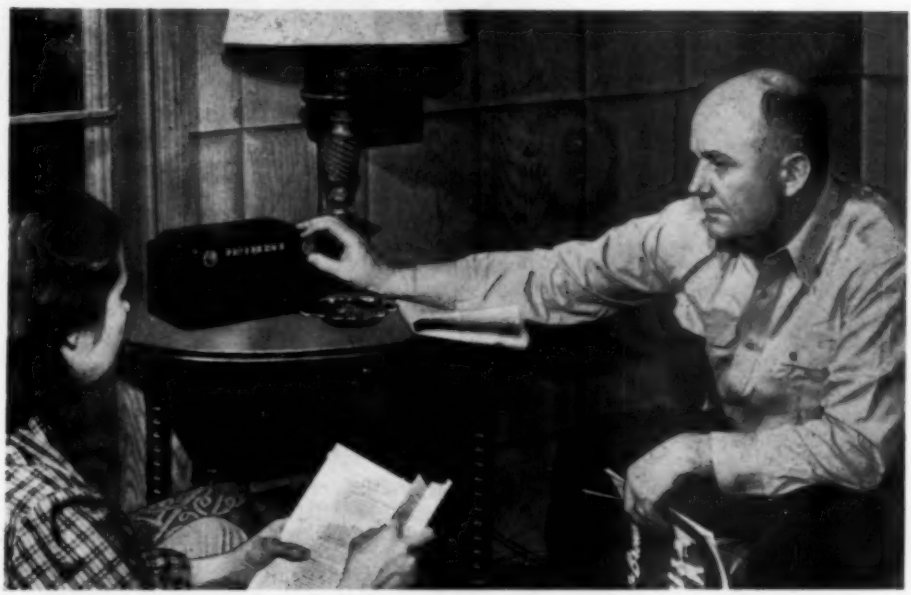

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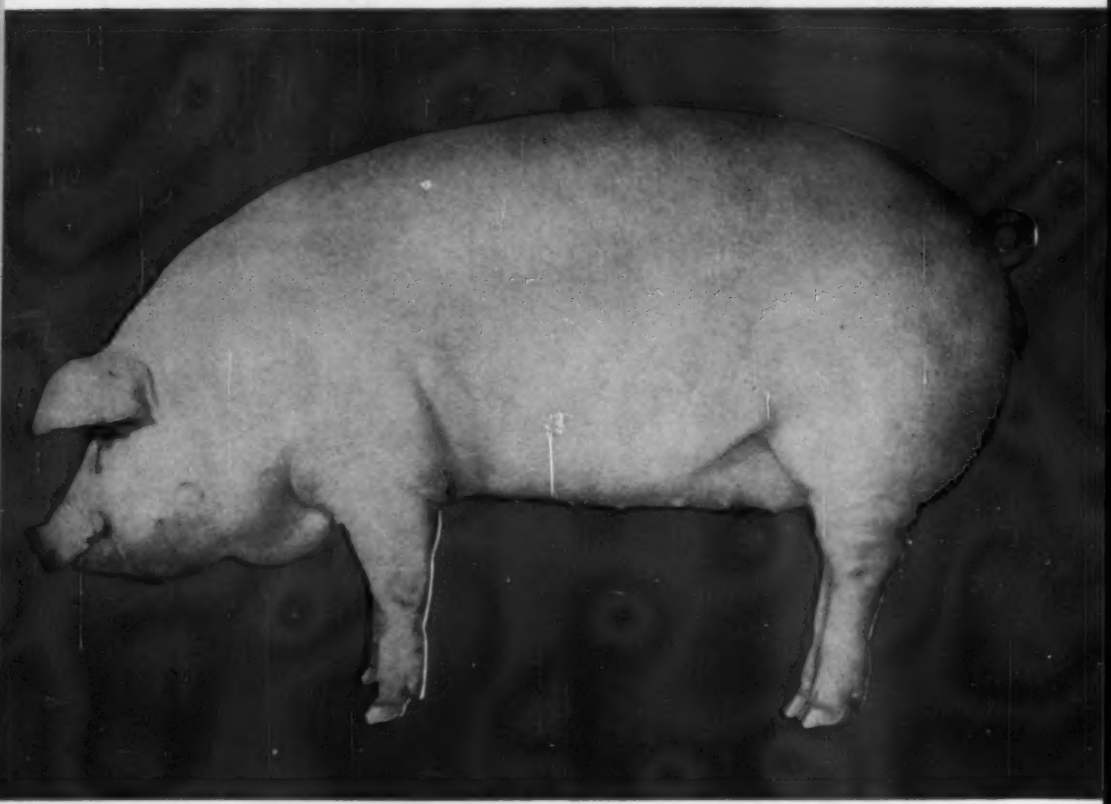

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Sequelae of Leptospirosis in Horses on a Small Farm

S. J. ROBERTS, D.V.M., M.S.
Ithaca, New York

IN 1952, AN OUTBREAK of leptospirosis occurred in horses on a small farm.[23] Of the 16 horses on this farm, 5 developed anorexia, elevated body temperatures of 104 to 105 F., depression, and icterus that lasted one to three days. A sixth, an 11-year-old mare, had no signs of illness but foaled two weeks prematurely and the foal died in two days, with marked icterus. The cause of death was not determined. Since the possibility of neonatal isoerythrolysis was eliminated, leptospirosis was strongly suspected.

The first 3 horses were affected late in March and showed a high, or rising, titer to *Leptospira pomona* two to three weeks later. The last 3 were affected in April and cultures of their blood during the acute illness were positive for *L. pomona;* their blood serum titers were negative, but rising high titers to *L. pomona* were found two to four weeks later. At the time of illness, their hemograms revealed a moderate leukocytosis and neutrophilia.

All 6 affected horses, except the mare that foaled prematurely, were treated with penicillin and streptomycin for several days during the acute illness. The other 10 horses on the farm showed no similar illness either before or after this time.

These cases were discussed in a 1955 report on leptospirosis in horses[4] and, for the past five years, further data, case histories, and results of regular serological tests have been assembled and are here reported.

RESULTS

The results of this study are graphically summarized (table 1).

Dr. Roberts is professor, Department of Medicine, Obstetrics and Ambulatory Clinic, New York State Veterinary College, Ithaca.

Five of the 6 horses that had a confirmed acute attack of leptospirosis subsequently were affected with periodic ophthalmia. The sixth, an aged mare (horse 6), was destroyed in September, 1952, because she had failed to conceive.

Horse 1, a 9-year-old mare, had a typical attack of periodic ophthalmia, with bilateral photophobia and lacrimation, on the second day of illness. After treatment with penicillin and streptomycin for three days, the eye condition had not improved so chlortetracycline was administered for the next three days. Within ten days, the condition affecting the eyes had greatly improved. Recovery was complete and the mare has had no recurrence of the ophthalmia in the past five years. Her high serum titer for *L. pomona* receded within a year to a low level, where it has remained. Her foal (horse 9), born soon after the acute attack of illness, was normal except for a high titer for *L. pomona* for several months. The titer receded to negative in one year and was still negative when the animal was sold as a 3-year-old.

Horse 2, 4 years old, remained on the farm as a brood mare. Her original high serum titer declined within one year to a low level, where it remained for two years. During the past two years, it increased to 1:1,000. This rise probably is not significant. On Feb. 2, 1954, she showed a slight keratitis in her left eye and, on June 11, about two years after the acute leptospirosis, a typical severe attack of periodic ophthalmia developed, involving mainly the left eye. Although treated promptly with chlortetracycline and later with streptomycin, she had repeated attacks during 1955 and 1956, resulting in the loss of sight in

the left eye and impaired vision in the right.

Horse 3, the mare which had the weak, icteric, premature foal, showed no acute illness but her complement-fixation titer was high 11 days after foaling and remained high until she was killed in February, 1955, because of blindness and failure to conceive. This mare had her first severe attack of periodic ophthalmia, in the left eye, in November, 1953, about 18 to 20 months after the attack of leptospirosis. During the previous summer and fall she was observed, on four occasions, to have a mild photophobia, miosis, and lacrimation of the left eye. Although antibiotic therapy with oxytetracycline and streptomycin and local treatment of the eye with atropine were administered, severe bilateral attacks of recurrent iridocyclitis occurred repeatedly during 1954, resulting in the loss of sight in both eyes.

Horse 4, a 2-year-old gelding, which at the time of its acute attack of leptospirosis had a negative titer for *L. pomona*, developed a definite, though not a high, titer three weeks later that persisted at this level until the horse was killed because of blindness in June, 1954. His first attack of periodic ophthalmia affected the left eye, on March 22, 1954, about 24 months after the acute illness due to *L. pomona*. Early treatment with streptomycin in large doses, together with local treatment with

TABLE I—Sequelae of an Outbreak of Leptospirosis (Leptospira Pomona) in Horses on a Small Farm, Including Yearly Blood Titers and Duration of Attacks of Periodic Ophthalmia

Horse No.	Age	1952 Mar.	1952 Apr.	1952 May	1952 Sept.	1953	1954	1955	1956	1957
1	9	▲ ■	+ CF 1:512	± CF 1:4	+ 1:2000	+ 1:800	± 1:100	± 1:100	+ 1:300	± 1:100
2	4	▲	+ CF 1:512	+ CF 1:32	+ 1:1000	± 1:100	1: ■	■	■	■
3	11	▲	+ CF 1:128	+ CF 1:64	+ 1:32000	+ 1:3 ■	+ ■	+ K ■		
4	2		C− ▲ CF	+ CF 1:16	+ 1:4000	+ 1:2000	+ K 1: ■			
5	1		C− ▲ CF	+ CF 1:32	+ 1:8000	+ 1:100 ■	■	■	K	
6	22		C− ▲ CF	+ CF 1:8	± K CF 1:4					
9	1 mo.		Born 4/1/52	+ CF 1:32	+ 1:1000	± 1:100	−	−	Sold Norm.	
11	1			−	−	−	−	−	−	−
13	24			−	−	−	−	−	−	−
14	4			± CF 1:4	± 1:100	−	−	−	−	± 1:100
16	4			+ CF 1:32	+ 1:8000	+ 1:8000	+ 1:8000	+ 1:1000	+ 1:10000	+ 1:10000
A*	18		○ − ▲ ▲	+ CF 1:16	+ 1:4000	+ 1:8000	+ 1:8000	+ 1:10000	+ K 1:30000	

▲ Period of acute illness; C Cultured *L. POMONA* from blood during illness; K Killed; CF Complement Fixation test; * Experimental mare; ○ Inoculated with blood from No. 6 taken during illness; ■ Duration of attacks of periodic ophthalmia.

The numbers assigned the horses above are the same as the numbers assigned to the horses in table 1 in the 1952 report.[22] Horses 7, 8, 10, 12, and 15 were noninfected horses with negative blood titers for L. pomona in 1952 whose blood remained negative until destroyed or sold; horse 7 was destroyed because of lameness in late 1952. Horses 8, 10, 12, and 15 were normal and were sold in the fall of 1952 with the exception of the last, which was sold in 1954. These horses were omitted from the table for the sake of brevity. The experimental mare A is the same mare as cited in table 2 of the 1952 report[22] and by Bryans (p. 38).[4]

atropine and cortisone, failed to prevent severe recurrent bilateral attacks in April and June that resulted in a marked loss of vision.

Horse 5, a 1-year-old gelding, had a negative titer for *L. pomona* when ill with leptospirosis, but developed a fairly high titer within three weeks. This titer dropped to 1:1,000 in 1953 and remained at 1:200 to 1:300 during 1954 and 1955. A mild but definite attack of periodic ophthalmia occurred Dec. 12, 1953, 20 months after the acute attack of leptospirosis, followed by two to three attacks with progressive severity and loss of vision in 1954 and 1955. It was destroyed in March, 1956. This horse was also treated during several of its acute attacks of recurrent iridocyclitis with antibiotics, corticoids, and local application of atropine and antibiotic ointments.

Horse 9, the foal of horse 1, was the only animal in this group which showed a fairly high titer immediately after the outbreak of leptospirosis, then completely lost its serum titer to *L. pomona* (in 1953, 1:100 in April and negative in June). This animal was sold in 1956, with no history of ophthalmia and with normal eyes.

Horses 11 and 13 remained on the farm, developed no titers for leptospirosis, and are normal.

Horses 14 and 16, that were on the farm during the outbreak of leptospirosis, were moved in the fall of 1952 to another small farm where about 10 horses of all ages were maintained. These 2 brood mares and the other horses on this farm have remained normal, with no signs of ophthalmia. One mare (horse 14) has an occasional low or questionable titer for *L. pomona*, while the other (horse 16) has had a consistently high blood titer for the past five years.

Horse A, an aged mare experimentally infected with *L. pomona* April 13, 1952, developed a typical acute attack of leptospirosis 11 days later, followed by the development of a moderately high blood titer. Six months later, she was used in sensitizing experiments and was given repeated doses of *L. pomona* by various routes over a period of 15 to 17 months.[4] Although her serum titer for *L. pomona* remained high, no attacks of periodic ophthalmia developed and when she was killed in November, 1956, her eyes were apparently normal except for a slight adhesion of the iris to the anterior capsule of the lens of the left eye. This adhesion may have been caused by anterior chamber paracentesis that was performed several times in 1953 and 1954.

No other horses on the farm, except those described, have developed periodic ophthalmia in the past five years. However, a filly, foaled in 1954 out of mare 3, was sold in the spring of 1956 and developed periodic ophthalmia in the fall of 1956.

DISCUSSION

In recent years, there has been increasing amount of evidence linking leptospirosis and periodic ophthalmia in horses. In the herd of horses discussed, the close relationship between affected horses and subsequent attacks of periodic ophthalmia appears significant. In a 1950 report,[18] of 3 horses that were febrile, jaundiced, and weak, 2 showed signs of ophthalmia at the time of the acute illness. Later the serums of these horses reacted positively to *L. pomona*.

In a 1951 report[17] on 4 horses that were febrile, weak, icteric, depressed, and exhibited anorexia for two to seven days, all 4 had antibodies for Leptospira when tested several weeks or months later. The one tested at the time of acute illness was negative. Three of these 4 horses developed typical periodic ophthalmia, with recurrent attacks two to four months after the initial illness.

In a 1953 report,[16] a horse that showed a fever, icterus, depression, and anorexia which lasted three days, had a typical bilateral iridocyclitis during this illness but recovered completely after treatment with penicillin and streptomycin. The blood titers for *L. pomona* increased from 1:500, on the third day of illness, to 1:100,000 four days later.

In a 1956 report,[21] 5 horses with leptospirosis due to *L. pomona* all exhibited a fever, icterus, depression, and photophobia, but only 2 had eye lesions resembling those of periodic ophthalmia at the time of the acute illness. These previous reports, together with this present one, tend to indicate that periodic ophthalmia may follow the usual fairly mild to moderate illness characteristic of leptospirosis in horses. In the case of horse 1 in this report, as in others cited,[16,18,21] signs of ophthalmia or iridocyclitis may have occurred at the time of the initial attack of leptospirosis. This is also true in human leptospiral infections.[15]

In the case of horses 2, 3, 4, and 5 in this report, and in 3 of 4 horses in another report,[17] the onset of definite signs of periodic ophthalmia occurred 12 to 24 months and two to four months, respectively, after the initial febrile illness. All of these cases of periodic ophthalmia were associated with positive blood titers for *L. pomona*.

This six-year study of a group of horses showed that once a horse is exposed and infected with Leptospira and exhibits a blood titer, the positive titer remains (with few exceptions, e.g., foal 9), as it does in cattle,[22] for six years or probably the lifetime of the horse. It is highly probable that the titer in foal 9 was due to passive transfer of antibodies from the recently infected dam to the foal by means of the colostrum, as has been demonstrated in calves.[6] High titers in horses persist whether or not periodic ophthalmia develops. Low and inconstant titers such as exhibited by horse 14 may be due to variations in the agglutination-lysis test itself or to the presence of nonspecific agglutinins in the mare's serum.

One of the first reports on equine leptospirosis[10] showed that 7.5 per cent of 291 normal horses and 85 per cent of 263 horses affected with recurrent iridocyclitis had leptospiral agglutinating antibodies in their serums. In 1950, it was reported[30] that of 86 normal horses, 37 per cent had agglutination-lysis titers in their serums of less than 1:300 and 12 per cent had titers greater than 1:300; while in 35 horses affected with periodic ophthalmia or recurrent iridocyclitis, all had antibodies to some degree, 6 per cent had a titer of less than 1:300, and 94 per cent had a titer greater than 1:300.

Other reports[4,17] and the data reported in this paper confirm this relationship between the serum titer and the disease. All 5 of the horses affected with periodic ophthalmia had positive blood titers for *L. pomona*. One of the titers was high (1:32,000) while another was low (1:200 to 1:300; table 1). The author has also found this close relationship in other affected horses. The titer level is apparently not related to the severity or course of the periodic ophthalmia.[30]

The agglutination-lysis test for leptospirosis may be useful in diagnosing periodic ophthalmia in horses with questionable histories or eye lesions. If the serum is negative, then the horse is probably not affected with periodic ophthalmia; if it is positive, then the condition may be periodic ophthalmia since some apparently normal animals may carry positive titers for leptospirosis. However, some[5] have definite reservations concerning the use of this serological test for the diagnosis of periodic ophthalmia.

Although most of the above reports indicate that *L. pomona* is the principal causative agent for leptospirosis, and possibly for periodic ophthalmia in horses, serological tests indicate that *Leptospira icterohaemorrhagiae* and *Leptospira canicola* may also infect horses.[4] In one report,[19] of 64 horses with periodic ophthalmia, 40 had high titers in their blood serum for *Leptospira grippotyphosa*, 13 for *L. icterohaemorrhagiae*, 6 for *L. pomona*, and 5 for *L. canicola*.

Many treatments for periodic ophthalmia or recurrent iridocyclitis have been advocated in the past, but with few satisfactory recoveries. Two older treatments that should probably be retained are: (1) keeping the affected horse in a darkened stall, and (2) the local use of atropine to dilate the pupil and prevent possible adhesions between the iris and lens.

In cases treated by the author in recent years, antibiotics and the corticoid hormones administered systemically seemed to have a mild alleviating and beneficial effect. The use of streptomycin and broad-range antibiotics has been recommended but they have given poor results in most instances. Horse 1 in this report, and another in which the iridocyclitis accompanied the initial febrile illness,[16] made lasting recoveries after the use of antibiotics. Such a response following treatment during an initial attack has been observed by the author in one other instance, and has also been reported,[7] but treatment with antibiotics during recurring attacks has given poor results.

Antibiotic therapy has also been of little value in treating leptospirosis in human beings;[8,15,25] however, there are indications that if used early they might be of value.[25] Streptomycin and the broad-range antibiotics were reported to be useful in treating and preventing or eliminating the carrier state in leptospirosis of cattle and swine.[20]

There may be several forms or causes of ophthalmia associated with leptospirosis, one associated with the initial acute attack

and another with recurrent attacks. The former appears to respond to antibiotic therapy; the latter does not. Since some have failed to recover Leptospira from the eyes of horses affected with recurrent attacks of periodic ophthalmia,[4,29] it has been suggested[2,4,27] that this might be an allergic condition. More study on the etiological factors of periodic ophthalmia or recurrent iridocyclitis must be completed before a satisfactory treatment can be recommended.

It is of interest that ophthalmia is a common complication following attacks of leptospirosis in man,[1,15,26] with ocular lesions occurring in as high as 44 per cent of affected persons two weeks to one year after the acute illness. During the acute illness, conjunctival injection and photophobia are common, with iridocyclitis and uveitis developing later. The recurrent type of iridocyclitis in man is rare but may result in loss of vision. In human cases of leptospirosis showing secondary ocular lesions, the prognosis is good in contrast to the poor prognosis in recurrent iridocyclitis in horses.

An interesting observation in leptospirosis in man was that when chemotherapy was administered early in the acute disease, the agglutination-lysis titers that developed subsequently were lower than in persons in which chemotherapy was started later in the course of the disease.

Horses 3, 16, and A (table 1) had high leptospiral titers and no antibiotics were administered, while horses 1, 2, 4, and 5 had relatively low titers and were given antibiotics early during the acute illness. This did not prevent the subsequent development of periodic ophthalmia in horses 1, 2, and 4 but the antibiotic therapy may have prolonged the interval (12 to 24 months) between the acute attack of leptospirosis and the first severe attack of periodic ophthalmia.

It is of interest to speculate that possibly, if high doses of antibiotics for a longer period of time had been employed in animals 2, 4, and 5, as was done in 1, the blood titers might have been even lower and possibly periodic ophthalmia might not have developed.

In 1946 and 1949, it was reported[13,14] that the feeding of 40 mg. of riboflavin daily in the grain was an effective means of controlling periodic ophthalmia in horses. The owner of the herd of horses described here was in the feed manufacturing business and, having had a few horses with the disease prior to 1952, started supplementing his grain with the recommended amounts of riboflavin in 1949. At least 40 mg. of riboflavin daily has been fed to these horses continuously, from 1949 to 1958, except during the pasture season when grain was not required, without controlling or preventing periodic ophthalmia.

More research must be done to satisfactorily explain the relationship between leptospirosis and periodic ophthalmia in horses. One author[11] reported success in producing periodic ophthalmia in 2 foals, but his experiment was not well planned. Another inoculated 10 young horses with *L. pomona* and no ophthalmitis was observed during a ten-month period after the acute infection.[5] He was unsuccessful in other attempts[4] to reproduce experimental cases of periodic ophthalmia.

One author[29] reported leptospiruria for several months after the acute illness, but this was not confirmed by others.[4] The role of horses as chronic carriers of *L. pomona* was considered of little importance.[4] The possibility of leptospirosis causing abortions in mares, as commonly occurs following leptospirosis in cattle and swine, requires further study. It is certainly not a common cause of abortion in mares, but some clinical reports[12,16,23] indicate this possibility.

Because of the apparent close association of leptospirosis and periodic opthalmia in the horses on this farm, they will be vaccinated with a leptospiral bacterin every February before the pasture season. Since a satisfactory immunity apparently develops after vaccination and persists for at least six months,[3,24] this practice should provide satisfactory protection for the spring, summer, and fall months when exposure to *L. pomona* is most likely to occur.

CONCLUSIONS

1) Clinical and serological evidence indicate that leptospirosis and periodic ophthalmia (recurrent iridocyclitis) in horses are closely associated.

2) Confirmed leptospirosis occurred in 6 of 16 horses, in 1952, and was followed by periodic ophthalmia in 5 of the 6 affected animals, the sixth horse being killed soon after the attack. Four of the 5 remaining horses developed periodic ophthalmia 12 to

24 months after the acute illness; the other animal developed an iridocyclitis 24 to 48 hours after the onset of the acute illness.

3) This six-year study indicates that once a horse is exposed to *Leptospira pomona* it develops a blood titer that, with few exceptions, persists for many years or for the lifetime of the animal.

4) The agglutination-lysis test for leptospirosis may be of diagnostic value in determining whether a suspicious ocular lesion is periodic ophthalmia.

5) Treatment of horses with recurring cases of periodic ophthalmia, with various antibiotics, was unsuccessful. However, antibiotic treatment of iridocyclitis associated with the acute illness caused by *L. pomona* seemed of value.

6) Routine feeding of at least 40 mg. of riboflavin daily to this group of horses did not control or prevent the occurrence of periodic ophthalmia.

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Leptospirosis and Periodic Ophthalmia in Horses

In 1953 and 1954, in Romania, leptospirosis was diagnosed clinically in man, cattle, and swine, and serologically in horses.

It was diagnosed clinically in horses, in 1956, and complete studies were made on 20 horses with periodic ophthalmia. In 3, a close parallel was found between antibody titer and development of iridocyclitis lesions. Of a total of 171 horses with periodic ophthalmia, 71 per cent had titers up to 1:102,400 whereas, of 535 healthy horses from the same areas, 15.7 per cent had titers up to 1:204,800.

In one area where the epizootic was chronic, 41 per cent of the horses had titers up to 1:51,200 whereas, in an area where there were acute cases of periodic ophthalmia, 85 per cent of the horses had titers up to 1:102,400.

The study indicates a close relationship between *Leptospira pomona* infection and periodic ophthalmia in horses.—*P. Jivoin et al. in Yearbook Inst. Anim. Path., 7 (1957): 46.*

Leptospirosis in Horses in Romania

In 1956, 10 cases of spontaneous clinical leptospirosis in horses were studied. They occurred in horses pastured on hills as well as in low swampy areas. The microagglutination-lysis test was positive eight to nine days after the onset of illness, with maximum titers of 1:51,200 to 1:409,600. After six to eight months, the titers had subsided to 1:800 to 1:3,200.

Similar tests on 101 clinically healthy horses, in three areas where cattle and horses were affected, showed 6.12 to 50.0 per cent of these groups with titers of 1:800 to 1:102,400, indicating subclinical infection. Experimental intravenous infection of 6 colts, 1 to 2½ years old, resulted in 2 of the 3 given *Leptospira pomona*, isolated from field cases, developing clinical infections (1 acute case after 14 days, and 1 case of bilateral ophthalmia after 18 days).

The third colt and the 3 given a mixture of *L. pomona* and *Leptospira canicola* remained well but developed agglutinins for the homologous organisms.

No etiological relationship was found between leptospirosis and infectious equine anemia.—*P. Jivoin et al. in Yearbook, Inst. Anim. Path. and Hyg., Bucharest, 7, (1957): 65.*

Leptospirosis in Horses.—Of 25 cases of leptospirosis in horses, near Zagreb, Yugoslavia, *Leptospira pomona* was isolated from 24, and *Leptospira sejroe* from 1—"the first time *L. sejroe* has been proved in the horse." Because of the prevalence of the disease in horses, prophylactic measures are recommended.—*Schweiz. Arch. f. Tierheilk. (March, 1958): 163.*

Leptospiras in Udders of Cows.—In leptospirosis of adult cattle, hemoglobinuria is rare, the most characteristic sign being a sudden agalactia immediately after the febrile period. Cows usually recover spontaneously in a few days. The syndrome was produced experimentally in 1 cow and leptospiras were found in her milk for only a few days but in the urine for a considerable period. The udder may be freed of organisms more quickly by a parenteral administration of penicillin.—*Mag. allatorv. Lap. (Feb., 1958): 33.*

Tuberculosis in Animals

Tuberculosis in cattle has been reduced from 5.0 per cent in animals tested in 1917 to 0.15 per cent in 1956. The percentage of slaughtered swine showing lesions has declined from 15.0 per cent in 1924 to 4.0 per cent in 1954, and 3.0 per cent in 1956. Most of the tuberculosis in swine is due to the avian-type bacillus. The bovine type has practically disappeared but in 1956, when 18 of 373 slaughtered swine were condemned and their origin was traced, 21 of the 28 cattle, on the farm of origin, reacted to the tuberculin test and 5 had extensive lesions. The 18 breeding swine on the farm all reacted to the test. The human-type bacillus has not been identified in swine in recent years, probably due to the prohibition against feeding raw garbage, especially that from hospitals or sanatoriums.

Tuberculosis in poultry has been reduced by improved management practices but remains a problem in the north central states where laying hens are retained for more than a year. The disease is found in wild birds in many parts of the country. Pheasants and turkeys are quite susceptible while ducks, geese, and swans are more resistant. Lesions are frequently found in pigeons and are reported to be common in starlings in England.

Tuberculosis is rare in dogs. Cats are resistant to the human type but are relatively susceptible to the bovine type of infection. Parrots in captivity seem more susceptible to the mammalian than to the avian bacillus and may be infected with the human type.

While tuberculosis has not been found in monkeys living in the wild state, in captivity they are the most susceptible of all animals. Foxes and mink raised in captivity are susceptible to the bovine type and mink are susceptible to the avian type of infection. There are 8 recorded cases of fatal tuberculosis in elephants, all of which yielded the human-type bacillus.—*J. H. Steele and A. F. Ranney in Am. Rev. of Tuberc. (June, 1958): 908.*

Laboratory Report on Leptospirosis in Georgia—1957

MAURICE W. HALE, V.M.D.

Tifton, Georgia

THE PURPOSE of this paper is to present the results of serological examinations of animal serums for leptospirosis during 1957. It may also serve to provide information concerning the incidence of various serotypes in this area.

MATERIALS AND METHODS

Clotted blood or serum specimens were submitted by Georgia veterinarians to the laboratory for examination.

Preparation of Control Serums.—Positive control serums for each serotype were prepared by the intravenous injection of living cultures in Stuart's medium into normal rabbits. Injections of 1, 2, 4, and 6 ml. were made at seven-day intervals. Seven days after the last injection, blood samples were taken and the serums harvested.

Negative control serums were obtained from normal healthy swine and cattle and preserved by addition of equal quantities of glycerin.

Preparation of Antigens.—Cultures of all serotypes were maintained in either Stuart's medium or Schuffner's modification of Verwoort's medium. Cultures were transferred at intervals of two weeks, or more frequently if required. Only young, vigorously growing cultures were used as antigens.

Micro-Agglutination Test.—Serial fourfold dilutions of each serum were prepared, using physiological saline solution. These provided final dilutions of 1:100 through 1:6,400. Five series of four dilutions each were prepared to permit the use of five leptospiral antigens. To 0.2 ml. of each serum dilution was added an equal quantity of antigen. The tubes were shaken and incubated at 30 C. for two hours.

A small drop of each mixture of diluted serum and antigen was transferred to a clean glass slide by means of a small inoculating loop. The drops were examined microscopically using a dry dark field condenser, a Pv 10/0.25 achromatic objective, and 10x wide-field eyepieces. Positive agglutination was determined by the formation of characteristic bright white spheres or lacelike networks in which the free ends of leptospiras could be seen moving. Definite evidence of agglutination in a serum dilution of 1:100, or higher, was considered a positive reaction.

RESULTS

During 1957, a total of 1,394 cattle and 480 swine (table 1) were tested. Of 3 dogs tested, 2 were positive to *Leptospira canicola* and 1 was negative. Of 3 horses tested, 2 were negative and 1 was positive to *Leptospira pomona*.

DISCUSSION

The data would indicate that *L. pomona* is the most prevalent serotype of leptospiras affecting swine and that *Leptospira sejroe* is the principal offending serotype in cattle. The incidence of leptospirosis in Georgia livestock can not be accurately estimated from this data because many of the specimens were from animals suspected of having the disease. Positive serological evidence of the disease was obtained from 33 counties, suggesting that the disease is widely distributed in Georgia.

TABLE 1—Leptospiral Agglutinins in Cattle and Swine in Georgia—1957

	Serotypes	Animal	No.	Percentage
Negative	.....	Cattle	437	31%
	.....	Swine	355	74%
Positive	Leptospira pomona	Cattle	338	24.0%
		Swine	113	24.0%
	Leptospira sejroe	Cattle	602	43.0%
		Swine	1	0.2%
	Leptospira autumnalis	Cattle	1	0.07%
		Swine	8	1.7%
	Leptospira grippotyphosa	Cattle	3	0.21%
		Swine	2	0.4%
	Leptospira ballum	Cattle	13	0.95%
		Swine	1	0.2%

The micro-agglutination test,[3] as used, is believed to be the most sensitive and specific test for leptospirosis. The complement-fixation test has been found to be genus specific, but not type specific. Plate and tube tests using killed antigens have been found lacking in sensitivity. While the micro-agglutination technique is laborious and time-consuming, it is still the method of choice in WHO control laboratories.

In cases where a single serum produces positive agglutination with more than one serotype, it is customary to designate the strain producing agglutination in the high-

From the Department of Animal Diseases, University of Georgia College of Agriculture Experiment Stations, Tifton.

The author thanks Delano Deen, Emilie McNeese, and Mary Walton for technical assistance.

est dilution as the offending serotype. This interpretation may not always be valid; a strain of *L. pomona* was isolated from a dog showing agglutination to *Leptospira autumnalis* in a higher dilution than to *L. pomona*.[1]

The histories of some herds of cattle with positive serological findings indicate that the disease has infected large numbers of animals without producing prominent clinical signs. Other herds with similar serological findings have been affected with abortions, mastitis, general morbidity, and even mortality.

The reason for this wide variation in pathogenicity is unknown. No information is available concerning the pathogenicity of serotypes other than *L. pomona* for cattle and swine. It has been shown that leptospirosis can be produced experimentally in calves by *L. sejroe*.[2] The finding that 43 per cent of the cattle tested in Georgia react to this serotype is evidence that the disease is present, but provides no indication of the degree of pathogenicity.

SUMMARY

During 1957, micro-agglutination tests for leptospirosis were made on the serums of 1,394 cattle, 480 swine, 3 dogs, and 3 horses in Georgia. Five leptospiral antigens were employed in fourfold dilutions of serum from 1:100 to 1:6,400.

The predominating serotype encountered in swine was *Leptospira pomona*, while *Leptospira sejroe* provided the largest number of reactions in cattle. Agglutinins against *Leptospira autumnalis*, *Leptospira grippotyphosa*, and *Leptospira ballum* were encountered in both cattle and swine in small numbers. Reactions to *Leptospira canicola* were obtained in 2 dogs, and 1 horse was positive to *L. pomona*.

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Research with Viruses

Viruses are all around and within us. It is the exceptional virus that causes disease; some may even help keep us healthy. Viruses grow and reproduce only within the living cell, being inert particles when not in cells. They can reproduce only by utilizing complex structures within cells. They are usually cell type-specific.

Each body cell contains nucleic acid, a complex chemical which directs the growth of the cell. A virus also contains nucleic acid and, when it invades a cell, the virus's acid assumes control of the cell's life. The cell becomes a virus-producing factory and, as each virus particle is liberated, it may infect another cell, resulting in a biological chain reaction. Often the infected cells are destroyed, as in paralytic poliomyelitis where paralysis results when the brain can no longer send messages to the muscles.

The study of viruses has been greatly enhanced since it has become possible to grow them in living cell cultures. In the past decade, dozens of new viruses have been discovered. Whereas formerly the study started with the disease, with an attempt to learn its cause, studies now start with the finding of a virus and an attempt is made to find the diseases with which it is associated.

Drugs are relatively ineffective against viruses since they can not kill viruses without killing host cells. To be effective, therapeutic agents must either prevent the virus from entering the cell by changing a cell wall; prevent the virus from controlling the cell after it enters; or prevent the new generation of the virus from escaping from the cell.—*Canad. J. Pub. Health (May, 1958): 196.*

Modified Viral Vaccine for Equine Respiratory Infection.—Of the several viruses associated with respiratory infections in horses, two have invaded fetuses and caused abortion. They have been passaged through hamsters at the Department of Animal Pathology, University of Kentucky and, when used as a vaccine, have stimulated resistance in a limited number of horses tested. A wide-scale field test of vaccine is planned.—*Blood Horse (June 21, 1958): 1301.*

Comparison of Epizootic Diarrhea of Suckling Rats and a Similar Condition in Mice

HENRY L. FOSTER, D.V.M.

North Wilmington, Massachusetts

EPIZOOTIC DIARRHEA of suckling mice has become a serious problem in recent years.[3] Losses in some colonies have been so high that production of mice has been slowed, or even halted, for weeks. In addition, the increasing use of suckling mice for virological studies has made the disease of major importance to research workers.

EPIZOOTIC DIARRHEA IN MICE

The diarrhea occurs in the second or third week of life and is characterized by a sudden onset, with a passage of large amounts of watery feces which ultimately soil the coat. In mild cases, the feces will dry and adhere to the underside of the tail. Some mice die in two days. Survivors may develop obstipation which, if unrelieved, may be fatal. Perianal necrosis and sloughing are sometimes found in recovered mice.

The outcome for individual litters has varied from 100 per cent mortality to 100 per cent recovery. In 1934, workers[8] found pathological changes limited to the ileum and colon, *i.e.*, enteric inflammation, ulcers, and sloughing. In 1947, others[6] described intranuclear inclusion bodies in the intestinal epithelium but no inflammation of the intestines. Subsequently, cytoplasmic inclusion bodies were found in the epitheliums of the small intestines in affected mice in a different colony, but intranuclear inclusion bodies were not seen and again enteric inflammation was absent.[7]

Although the available evidence indicated an infectious origin, all attempts to identify any specific bacterial or viral agent have been unsuccessful.[5] Nevertheless, the disease was considered to have been transmitted, resulting in the occurrence of inclusion bodies between the tenth and fifteenth day, by feeding unfiltered, diarrheal feces to suckling mice.[7]

To add to the confusion, the occurrence of yet other cytoplasmic inclusion bodies in the epithelium of the small intestines of mice without diarrhea was also described,[7] yet the relationship of the first reported type of cytoplasmic inclusion body to the disease could hardly be doubted. The cytoplasmic inclusion bodies were believed unmistakable, even at low magnification.[4] They were extremely numerous, brilliantly stained, but obviously different from the features shown in the rat intestines (fig. 1, 2). It was suggested that there might be at least two different viral agents causing diarrheal epizootic disease, because of the difference in the lesions found. Clearly, several unrelated causes might nevertheless produce the same clinical picture.

Various factors have been considered by many workers as possible contributory ones, e.g., the season of the year, humidity in animal rooms, strain susceptibility, parity of mothers, size of litters, diet, and even the type of bedding. No proof has been consistently obtained to incriminate any one, or any combination, of these conditions.

In 1957 it was claimed[5] that, in experimental work with mice, a filterable agent was isolated. This will require confirmation because many workers (published and unpublished records) have failed to identify a virus or viruses. It is unfortunate that the work[5] did not include observations on pathological changes so that comparisons could be made with the well-known works by the various other authors cited in this paper.

EPIZOOTIC DIARRHEA IN RATS

Clinically, at least, an identical disorder occurs in suckling rats. Due to basic differences in the methods of breeding rats and mice, the disease may have been far less recognizable by the rat breeder, and for that reason has not been reported. Our experience is that the mortality is less in the suckling rat than in mice, but the disease still is of practical economic importance.

The syndrome occurs about nine to 13 days after birth, but most commonly between 11 and 13 days. Prior to any obvious intestinal upset, there is anorexia and loss

From The Charles River Breeding Laboratories, Inc., North Wilmington, Mass.

The author thanks Dr. J. R. M. Innes for his assistance in writing this paper.

of weight. The rats leave the mother's nest, become scattered in the cage, and obviously lose nourishment. The skin is chalk-colored and feels cold. Slight pressure on the abdomen causes watery stools to be extruded. In a few days, the rats once again take to the nest and suckle their dams. Because of the habit of piling on each other while asleep, the pups quickly become soiled. After the thirteenth day, the hair becomes matted and the stools are more firm. By the sixteenth day, the acute period is over and more than 90 per cent of the animals show sudden improvement, as if some effective therapeutic agent had been given. Like the condition in mice, many rats may show partial occlusion of the anus by caked feces.

Relief of the obstipation by therapeutic means is indicated. By 21 to 23 days of age, there is a complete recovery and the previously sick animals can not be differentiated from the pups of unaffected litters. There are no obvious sequelae and no deterioration in the ultimate growth and fertility of affected rats. In our experience, whenever 1 pup in a litter became ill, the whole litter was affected.

Some tentative preliminary attempts were made to transmit the disease to suckling rats by injection of a filtrate of intestinal contents, but with negative results. No pathogenic bacteria have been isolated from the feces.

Tissues from 16 affected and 8 healthy rats of the same age were studied. There were no unmistakable gross lesions in any of the thoracic or abdominal viscera, including the small or large intestines. Sections were cut from many levels of the alimentary tract. As in the case of the mice,[6,7] there was no hemorrhage, no necrosis of the surface epithelium, no ulcers, and no inflammatory reaction.

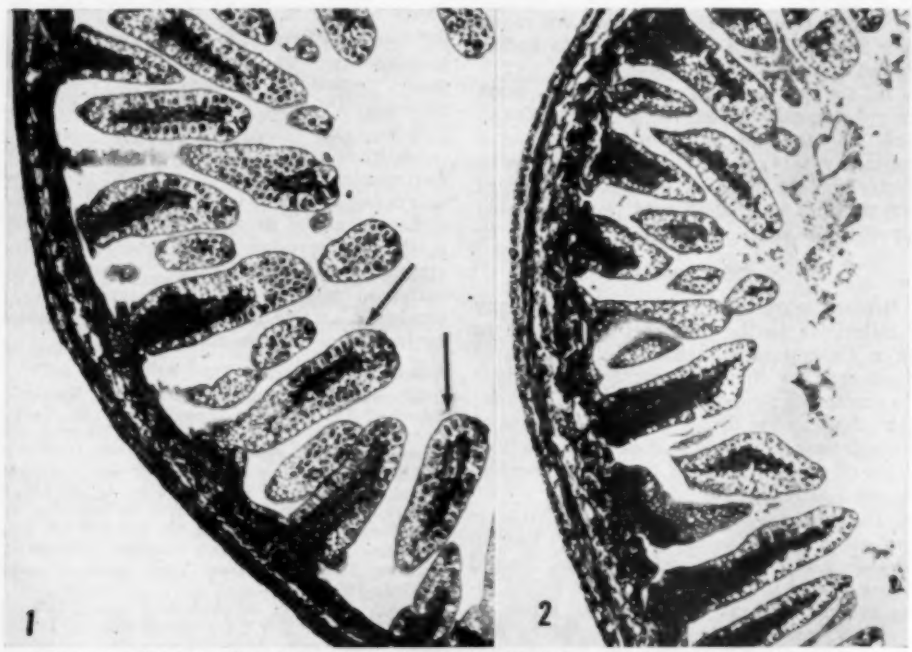

Fig. 1—Small intestine from a rat with diarrhea. Notice the absence of inflammatory reaction. Almost every epithelial cell contains a large, homogeneous, pink-stained globule surrounded by a clear space, the nucleus being pushed to the bottom of the cell. The "inclusion body" (arrows) lies in this space and is enormous compared with the cytoplasmic bodies described in mice. Many cells are empty, which may indicate that some of the bodies have fallen out during preparation of the sections. Compare with identical picture in intestinal epithelium from a normal control suckling rat (fig. 2). H & E stain; x 100.

Fig. 2—Small intestine from a nondiarrheic suckling rat. The same cytoplasmic bodies are present as in diseased rats, but there are more cells which appear empty of any material. H & E stain; x 100.

A careful survey of the sections of the intestines did not disclose the presence of either intranuclear or cytoplasmic inclusion bodies in the epithelium, as seen in mice.[7] In a few rats affected with the diarrhea, however, some curious changes were found and these are illustrated (fig. 1, 2) because it is doubtful if they have been described.

At some levels of the small intestine—never all, and never in the duodenum or large intestines—the epithelium was sometimes highly vacuolated, almost like a severe hydropic degeneration. In some sections, however, the cytoplasm of almost each cell was filled with a huge, homogeneous, hyaline-like, pink-stained "globule," usually surrounded by a clear space (fig. 1, 2). Consequently, the apparently hydropic changes (*i.e.*, without globules) may have been artifacts and due to the bodies dropping out in the preparation of the sections. The change might be a normal one in suckling animals; or it may be some cellular secretory product. The globules are many times larger than the cytoplasmic bodies found in mice with diarrhea.

It should be stressed that the same morphological appearances were found just as often in the intestines of the 8 normal rats examined. Examination, with histochemical tests, of a large number of normal and unaffected rats would possibly provide the solution.

DISCUSSION

Epizootic diarrhea in rats has many similarities to the disease in mice: There is a clear, unmistakable, clinical picture; there are no obvious gastroenteric lesions to account for the sudden onset of diarrhea; and there is the problematic significance of cytoplasmic inclusion bodies in the epithelium of certain parts of the small intestine in some affected rats.

There is no proof that the condition is infective although, superficially, it has all the characteristics of an infection. Should it be proved to be infective, there would remain the provocative problem of cross infection between mice and rats. Many different species have their own distinctive form of "infantile dysentery," e.g., children, calves, rats, and mice.

Nutritional factors would seem to be excluded from etiological considerations because in our colony the same ration has been fed for years. As in mice, many female rats with affected litters may be rebred and produce healthy young. Transmissibility of the disease by water can be excluded, for the condition has occurred in litters of breeding females which had been given sterile water. The disease has also occurred in animals when cages and water bottles were sterilized.

As in mice, high-producing rats seem to be more prone to the disease. We have no data to support the idea that the mother's milk is a factor controlling the onset of the diarrhea, but such a hypothesis can not yet be ruled out. Examination of the feces has not revealed the presence of protozoa which might be involved. The problem remains one worthy of detailed study.

SUMMARY

The clinicopathological features of epizootic diarrhea of suckling mice are compared with those seen in suckling rats. There appear to be no essential points of difference between the condition in mice and rats except that claims have been made regarding transmissibility of the condition in mice (*i.e.*, it is caused by a virus).

In the rat, as in the mouse, there are no gastroenteric lesions to account for the diarrhea, and no "viral" inclusion bodies either in nuclei or cytoplasm of intestinal epithelium have been unequivocally demonstrated. Some cytoplasmic "inclusions" illustrated might more likely be secretory products. Attempts to transmit the disease by intestinal filtrates failed.

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Purulent Keratoconjunctivitis in Laboratory Rats, Caused by *Micrococcus Pyogenes* var. *Aureus*

HENRY L. FOSTER, D.V.M.

North Wilmington, Massachusetts

An acute and subacute infectious ocular disease of rats, spread from rat to rat, and possibly by animal handlers, has been observed on different occasions and in a large number of young rats—never in adult animals. It occurs most frequently in recently weaned animals, but infection can arise during the lactation period and affect several in a litter.

It may affect one eye or both at the same time. The cornea initially becomes dry and soon loses its normal, moist, glistening state. An opaque dry film appears quickly and, in a day or two, pus may be profuse and the lids are soon adherent. There is no loss of physical condition. The infection is apparently limited to the eye.

Micrococcus pyogenes var. *aureus* was isolated from cultures from 5 rats. Other organisms isolated, e.g., *Streptococcus faecalis* and *Aerobacter aerogenes*, were probably contaminant or commensal organisms.

The 5 affected animals, which were examined bacteriologically, were killed and both eyes were fixed in formalin; sagittal sections through a median plane were cut and stained with hematoxylin-eosin and by the Giemsa method (for inclusion bodies).

All the animals presented a uniform picture of a process confined to the conjunctiva, cornea, and uveal tracts. There was no inflammation or pus in either the anterior or posterior chambers. The lens was normal. The conjunctiva was infiltrated with leukocytes and macrophages, and parts of the surface were covered by detritus, with raised plaques of stratified squamous epithelium, some of which were highly vacuolated. No inclusion bodies were present. The cornea was not ulcerated, but it was edematous. There was separation and disruption of the elastic fibers, and it was infiltrated with polymorphonuclear and eosinophilic leukocytes. There were no hemorrhages. At the junctions of the iris, ciliary body, cornea, and sclera, there was also some inflammatory reaction.

Treatment by one or two local applications of ophthalmic ointment containing chlortetracycline (Aureomycin*) resulted in a rapid and complete recovery.

Young rats should be carefully checked for the appearance of the eye condition, because it can easily spread, as long as they are kept in litters.

SUMMARY

Micrococcus pyogenes var. *aureus* has been isolated from each of 5 young weaned rats affected with purulent keratoconjunctivitis. The condition can be controlled by local application of ophthalmic ointment containing chlortetracycline (Aureomycin).

*Produced by American Cyanamid Co., Pearl River, N. Y.

From The Charles River Breeding Laboratories, Inc., North Wilmington, Mass.

The author thanks Dr. J. R. M. Innes for his assistance in writing this paper.

Use of Dental Films in Veterinary Practice

W. P. BLAKE, D.V.M.; J. MICUDA, D.V.M.; J. W. HUBBARD, D.V.M.

Phoenix, Arizona

For radiographing small areas, *i.e.*, the feet (fig. 1) or the oral cavities (fig. 2) of pet animals, dental film packs have proved easier to use than the average cassette. Since we do not consider ourselves highly proficient in the use of the x-ray machine, we will offer few suggestions as to the machine settings other than those we use.

Procedure.—The only equipment used, other than the x-ray machine, are dental film packs* and tape. These are used for radiographs of extremities of pups or small dogs (fig. 3). They are also useful in diagnosing the cause of lameness in large dogs such as Greyhounds (fig. 4). The owner usually holds the animal, thus limiting the number of exposures of staff members to x rays.

Sedation is seldom necessary as the film

Drs. Blake, Micuda, and Hubbard are small animal practitioners in Phoenix, Ariz.

*Kodak occlusal super speed film, $2.85 for box of ten packs, each in a paper holder.

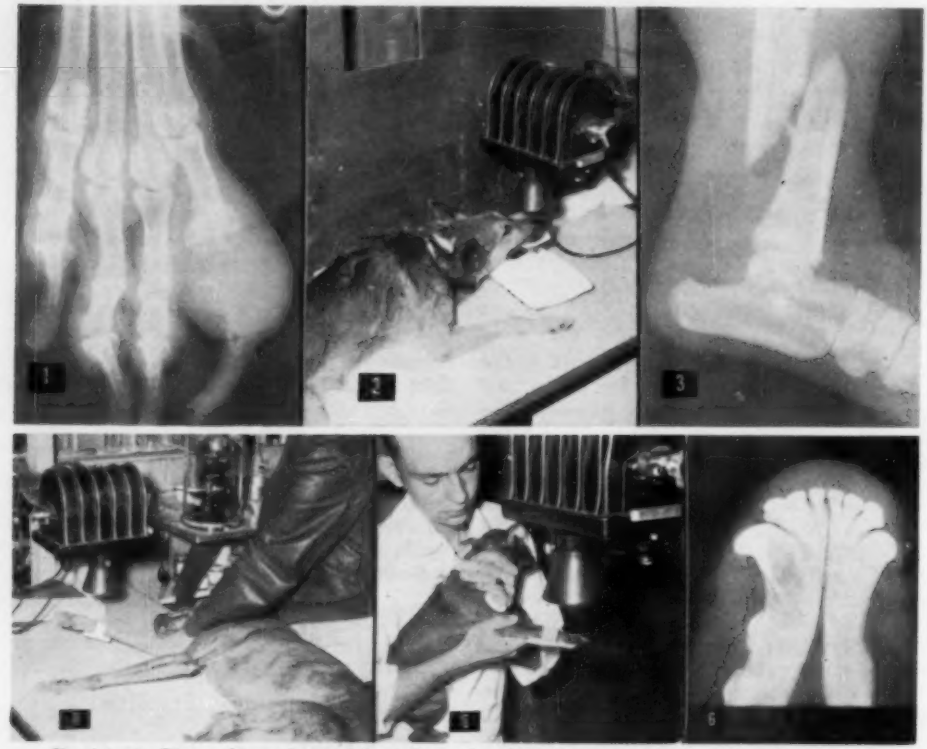


Fig. 1 to 6—Film packs can be used for radiography of small areas such as dog's foot (fig. 1); oral cavity (2, 6); dog's leg (3, 4, 5).

is taped to the area to be exposed, which is held about 6 inches from the head of the x-ray machine (fig. 5).

Our machine delivers 15 ma., and exposures between ½ and ¾ of a second produce good radiographs, depending on the thickness of the part. The films are developed in the same manner as regular films. All films have an indentation stamped in them when packed so, by placing the indentation on the right side when taking a picture, no error is made in reading the film.

Conclusions.—The advantages of these films are: (1) little blur from movement due to long exposure; (2) usually no sedation is necessary; (3) reduced exposure of the staff to repeated xrays; (4) films are packed in paper holders which contain two films, making it possible to give the duplicate to the owner; (5) clearer and sharper pictures are obtained than with the speed screen (fig. 6).

Dental film packs may also be used to check the amount of x-ray exposure received by employees, by simply fastening a film to their belts or shirts with paper clips. If the film, which is developed at the end of a week, is dark and the paper clip shows, the employee is receiving too much exposure.

Cross Immunity Between Canine Distemper and Rinderpest.—Six ferrets were inoculated subcutaneously with lapinized rinderpest virus and 20 days later each was inoculated with spleen from a ferret with distemper. Two of the 6 ferrets, and 2 controls, died from distemper; the rest remained healthy. Also, 3 cattle inoculated subcutaneously with 5 ml. of spleen suspension from a ferret with distemper resisted infection with 300,000 minimum lethal doses of virulent rinderpest virus.—*Vet. Bull. (May, 1958): Item 1454.*

Seasonal Fluctuations of Gastrointestinal Nematodes of Imported Beef Feeder Calves in Northeast Iowa

L. H. PETRI, Ph.D.

Waverly, Iowa

THIS PAPER reports a study of seasonal fluctuations of gastrointestinal nematodes, as determined by eggs per gram of feces, in 80 Hereford spring calves originally from Phillips County, South Dakota. The calves were about 6 months of age when received in November, 1956. They were kept in a feedlot in Bremer County, Iowa, for 11 months until they were marketed. They were given no anthelmintic treatment at any time.

Little is known about seasonal fluctuations of gastrointestinal helminths of cattle in Iowa. Seasonal fluctuations in populations of adult helminths of cattle in Australia—and their correlation with the season of birth of the calf, rainfall, and temperature—have been discussed in some detail.[8]

MATERIALS AND METHODS

The number of eggs per gram of feces was determined by a modified Lane's[2] technique, using a saturated sodium nitrate flotation medium. Each month, egg counts were made on 20 samples collected at random from fresh stools in the feedlot. Collections began in February, 1957, and continued through October when the cattle were marketed.

Eggs were identified according to genus as far as possible, based on morphological characteristics.[3,7] Eggs of the genera Nematodirus, Trichuris, Bunostomum, and Strongyloides can be recognized with certainty. The remaining genera were placed in the following groups: Haemonchus-Oesophagostomum—group 1, and Cooperia-Ostertagia-Trichostrongylus—group 2.[4]

RESULTS

The results of the counts as recorded (table 1) show the range of eggs per gram of feces for 20 samples and the mean count for the herd for each month. No samples yielded a negative egg count. The mean count, which in February was 71.9 eggs per gram, rose sharply during March and reached a peak of 188.1 in April. This was followed by a sharp decline during May and June, with a slight increase during the remaining months (graph 1).

The fluctuations of the various genera or groups during this study are shown (table 2). The sudden rise in number of eggs per gram in the early months of spring occurred almost exclusively in groups 1 and 2.

TABLE 1—Monthly Fecal Egg Counts of Nematode Eggs from Feedlot Calves 8 Months Old

Month (1957)	No. of samples taken	No. nematode eggs per gram of feces	
		Range	Mean
February	20	4 to 190	71.9
March	20	42 to 253	104.8
April	20	60 to 476	188.1
May	20	24 to 469	88.5
June	20	3 to 52	18.0
July	20	2 to 108	29.5
August	20	5 to 181	48.5
September	20	4 to 141	38.8
October	20	3 to 130	48.2

Group 3 (Nematodirus spp.) apparently had reached its peak at this time, or earlier. Nematodirus showed a gradual decline throughout this study and no eggs of this genus were found during September and October.

Groups 4 (Trichuris spp.) and 5 (Bunostomum spp.) followed the same trend as that of group 3. No Strongyloides were seen in this study.

DISCUSSION AND CONCLUSIONS

The method of determining the number of worms present by the number of eggs per gram of feces is admittedly imperfect. Nevertheless, it remains the most practical method yet devised of measuring worm burdens of animals. If this method is used on a herd basis, and if enough samples are taken over an extended period of time, some of the factors may be eliminated which might be significant if done on an individual animal basis with a limited number of samples.

The interpretation and significance of fecal egg counts is still controversial, especially as it pertains to cattle. It has been estimated[3,6] that a level of 300 mixed strongylate eggs per gram suggests borderline or subclinical infection. In the present study, only during the peak season (table 1) did the counts indicate such infection. This occurred in 10 per cent of the samples

Dr. Petri is professor of biology, Wartburg College, Waverly, Iowa.

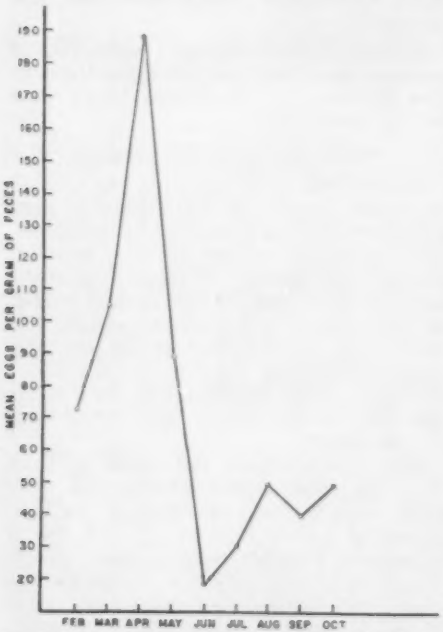
TABLE 2—Mean Differential Fecal Egg Counts on 20 Samples from Feedlot Calves 8 Months Old

Month (1957)	Group 1 Haemonchus and Oesophagostomum spp.	Group 2 Trichostrongylus, Cooperia, and Ostertagia spp.	Group 3 Nematodirus spp.	Group 4 Trichuris spp.	Group 5 Bunostomum spp.
February	12.6	34.6	21.2	3.0	0.2
March	14.5	81.2	5.7	2.7	0.3
April	36.8	140.4	3.1	3.1	0.1
May	15.6	70.8	1.5	0.45	0.05
June	2.1	16.0	0.05	0.25	0.0
July	2.3	24.8	0.035	0.05	0.05
August	8.6	39.4	0.1	0.05	0.0
September	4.3	34.5	0.0	0.0	0.0
October	3.8	21.4	0.0	0.0	0.0

collected in March and April, and 5 per cent in May.

An examination of table 1 and graph 1 reveals a seasonal fluctuation of gastrointestional nematodes in feeder calves imported from range land and kept under feedlot conditions. This could indicate that there is a strategic time for anthelmintic treatment if worms are to be prevented from reaching pathogenic numbers.

Evidently the sudden rise in April was due to reinfection in the feedlot of groups 1 and 2. The length of the prepatent period of most of the nematodes in these two groups would preclude this build-up in egg production being due to a worm burden present in November of the year before.



Graph 1—Monthly fluctuations of mean fecal egg counts in calves 8 to 17 months old.

In one study, cattle, at the age of about 18 months, developed a natural resistance which continued for the remainder of their lives.[8] This study seems to indicate that it would be wise to extend future studies over approximately 24 months to determine whether the peak production of worms is repeated every spring or whether the animal develops sufficient resistance to avoid such a repetition.

SUMMARY

In this study of 80 Hereford calves, imported after weaning, from range land of Phillips County, South Dakota, and maintained under feedlot conditions of northeastern Iowa, there was: (1) a seasonal fluctuation in gastrointestinal helminths, with egg production at a peak in early spring; (2) a worm burden in some calves (in early spring only) sufficient to suggest borderline cases of subclinical or pathogenic infections; and (3) evidence of a probable strategic time for worming cattle raised under these conditions.

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Clinical Views on Cesarotomy in Cattle

JURGEN LENSCH, Dr. med. vet.

Krempe/Holstein, Germany

CESAREAN SECTION in cattle has increased in importance as an obstetrical method during recent years. In the late 1930's, it was used with varying success. My own experience in this field is limited to its use in Germany and the United States. That the operation remained so long in the experimental phase in European countries is probably due to the following four causes: (1) the excellent refinement of embryotomy (fetotomy); (2) the low economic value of calves (about 50% below the present-day level); (3) the predominance of the horse; and (4) the unavailability of antibiotics.

In North America, the early development of the operation resulted from four causes: (1) the relatively high economic value of calves; (2) the tendency toward specialization which has resulted in experts in special fields; (3) the attractiveness of surgical methods to the American veterinarian, so impressive to every European visitor; and (4) a high standard of aseptic surgery and close cooperation with experts in human surgery, resulting especially in teamwork in publications.

In most cases where fetotomy is indicated, cesarotomy could be used as well; even with dead, decomposing fetuses, and with deformed fetuses, e.g., schistosomus reflexus.

TECHNIQUE

The discussion of operational methods is often difficult for me to understand. I should like to cite the reply of an American with extensive experience in this field when I asked him about the best operational method:

> I have carried out more than 500 cesarean sections in standing and lying cattle, as laparotomy right and left, in the lying animal in the median plane, paramedian left and right, according to the position of the fetus. Each of these methods has its drawbacks and advantages. In obstetrical surgery, different to all other surgery, it is of utmost importance to master every technique according to the position of the fetus. The obstetrical patient forces the veterinarian to adapt his method to the special condition.

Dr. Lensch studied at Cornell University, Ithaca, N.Y., as a Fulbright scholar.

The fastest cesarean sections are performed with the animal in a standing position. This can be done in any stable without special preparations and with only two persons assisting. But the method is dangerous and sometimes difficult, as the operation should be finished within an hour. If it lasts longer, the animals may lie down which makes the operation more difficult.

The operation I will describe would be indicated with a fetus too large for delivery and a uterus displaced toward the left side. Under these circumstances, a left flank operation, with the animal standing, can be performed in about 30 to 45 minutes.

Anesthesia.—Epidural, paravertebral, and subcutaneous anesthesia is combined. To prevent labor altogether, 8 to 10 cc. of a 2 per cent isocaine solution (procaine with adrenaline) is injected epidurally. For paravertebral anesthesia, 20 cc. of this solution is injected at the lateral intervertebral foramina of the last two thoracic and first two lumbar vertebrae. For subcutaneous anesthesia, solution is injected along the planned line of incision to achieve an immediate anesthesia of the cutis.

This additional anesthesia makes it possible to start the operation in five to ten minutes, compared with a waiting period of about 15 to 20 minutes after a general paravertebral anesthesia, which may be of importance in saving a calf of lowered viability.

Operation.—The incision begins about 10 cm. ventral to the transverse processes of the lumbar vertebrae and continues parallel to and 10 cm. from the last rib for 30 to 40 cm. After incising the cutis, the superficial and deep trunk fasciae are cut; then the oblique abdominal muscles and the fascia transversus abdominis. Only two small nerves are cut by the incision. In most cases, the bleeding is stopped by simple arterial pinching; ligatures are used only in exceptional cases.

After cutting the peritoneum, the rumen is pushed aside and the surgeon grasps the gravid uterine horn, drawing it as far as possible extraperitoneally, where an assistant holds it. As long an incision as pos-

sible is made in the uterus along the greater curvature of the gravid horn, without regard to caruncles. The fetal membranes are opened and the fetus is extracted by an assistant.

The uterus, still held by an assistant, is closed by a running suture (No. 5 catgut), starting from the cranial end of the incision and penetrating the serosa and muscularis but avoiding the mucosa so the fetal membranes will not be fixed to the uterine wall. Removal of the membranes after the extraction is not necessary as, in most cases (90%), they leave the uterus *per vagina* in about two to six hours after the operation.

Protruding parts of the membranes are excised so they will not interfere during suturing. If the fetus is living, a second line of sutures is not only unnecessary but contraindicated, as it may lead to additional tissue irritations. The strong disposition of the uterus to contract results in a quick sealing of the wound, thus guaranteeing a safe occlusion. In case of a delayed birth with an atonic and sometimes fragile uterus, a second line of sutures is required.

After extraction of a live fetus it is not necessary to insert chemotherapeutic agents into the uterine cavity as they would be washed out with the afterbirth. If the fetal membranes are still retained 24 hours after the operation, these agents are inserted and 5 or 10 cc. of ergotin are injected subcutaneously. If necessary, the afterbirth can be removed manually on the ninth day after the operation. Earlier removal (within the first three days) may be dangerous for two reasons: (1) injury to the uterine wound and danger of perforation; and (2) serious pains, resulting in reduced food consumption and milk production.

If the fetus is dead or infected, it is advisable to insert chemotherapeutic agents and to give a prophylatic injection of 50 cc. of "anti-gas-bacilli serum." The operation is completed by suturing the peritoneal inner abdominal muscles with a continuous catgut suture. A peritoneal suture without inclusion of the inner abdominal muscles can not withstand the pressure of the rumen and will often result in fissures and formation of adhesions of the viscera and the muscles.

Then the outer abdominal muscles are closed with catgut sutures, stitching through the inner and outer muscles at the same time in order to avoid the formation of cavities. In the case of fat animals, it seems necessary to stitch the subcutaneous fat tissue in an additional running suture, as this tissue has a tendency to heal poorly. The skin is closed by "intrapercutaneous" sutures with nylon thread.

Antibiotics can be used intraperitoneally, and also while suturing the skin incision, to avoid wound infection. Covering the wound with a paste or dressing is unnecessary. Only if the patient's temperature is over 40 C. (104 F.) is prophylactic application of antibiotics or sulfonamides indicated.

No special instructions concerning the treatment of the animal after the operation are necessary. The patient can be fed the same amount of concentrates and roughages as before parturition and, four to six hours after the operation, milking can be done by hand or by machine.

The nylon sutures can be extracted eight to ten days after the operation, and the patient can be bred 12 weeks after the operation.

PERSONAL EXPERIENCE

Since 1954, I have performed 84 cesarean sections on cows, under regular farm conditions, with one assistant. All of these operations—with two exceptions—were done on cows in a standing position. Of the 84 patients, 7 laid down during the operation, which was then continued with the animal in this position. Of the 66 living calves, 7 soon died or had to be killed and 11 were dead (2 were cases of schistosomus reflexus). Of the 84 dams, 72 showed normal appetites after the operation, and there was no disturbance of their general conditions and no deviation in milk yields, compared with those of cows which had given birth normally. The cows showed no signs of pain when hand-milked or machine-milked. The wound margins were sealed after three to five days and healing was *per primam*.

Of the remaining 12 patients, 4 had to be slaughtered within the first week after the operation (1 case of peritonitis; 1 of anaerobic infection; 1 of circulatory failure; and 1 of abdominal dropsy). In 8 animals, complications required one or more aftertreatments (1 case of retention of afterbirth because of an infected fetus; 1 of ascites of fetal membranes; 1 of anaerobic infection; 1 of peritonitis; 1 of wound infection; 1 of atonia of the in-

testines and watery diarrhea; 1 of tympany; 1 of simple retention of afterbirth).

SUMMARY

Of 84 cesarean sections in cattle, 85.7 per cent terminated in healing *per primam;* 9.5 per cent in healing *per secundam* (one or several after-treatments); and 4.8 per cent required slaughtering.

Eight cows, 2 of which had a torsion of the uterus and 1 with twins, had a retention of the afterbirth but only 2 resulted in complications. Of the 39 patients operated on in 1954 and 1955, 9 were no longer used in breeding but 30 were served or inseminated again, with 24 becoming pregnant. One cow aborted in the sixth month of pregnancy, 21 cows calved normally, and 2 required veterinary aid (1 extraction due to posterior position and 1 cesarean section because of an extremely big calf—128 lb.).

The pregnancy results of the 1956 to 1957 cases are not yet known.

Cesarotomy in the Mare—A Case Report

EDWARD C. MELBY, Jr., D.V.M.

Middlebury, Vermont

SINCE CESAREAN section in the mare is infrequently done, we believe that this one, done under unfavorable circumstances, should be added to reports of one in 1948,[1] and another in 1952.[2] In both instances, the facilities for surgery were more adequate than ours.

CASE REPORT

The patient was a 12-year-old Morgan mare that was believed to have previously foaled twice without difficulty. No breeding date was available. On May 5, 1957, the mare was depressed, wandered off to the far end of the pasture, and ate very little. Two days later, the udder was distended, milk dripped from the teats, and there was a sanguineous discharge from the vulva.

We were called to see her on the evening of May 9 when, for the first time, the mare was in labor. She was straining, restless, and stumbled when she walked. Her temperature was 104.4 F., pulse rate 99, respiratory rate 33, and the mucous membranes were injected. The vagina was dry and the cervix fully dilated. The foal, apparently dead, was in transverse position and dorsal presentation with its head in the right flank, the result of a bicornual pregnancy.

Although the prognosis was unfavorable, the owner decided to permit a cesarean operation. Surgery took place in a paddock lighted with automobile headlights and a floodlight.

Surgical Procedure.—Chloral hydrate-magnesium sulfate solution was administered intravenously until the proper plane of anesthesia was reached; more was given when required during the operation. Anesthesia was satisfactory except twice, when the mare recovered sufficiently to attempt to rise.

After the left flank was clipped, shaved, and disinfected, a 20-inch incision, from a point midway between the last rib and the external angle of the ilium, extending ventrally and slightly anteriorly, was made through the skin and muscles to the peritoneum. The large deposits of retroperitoneal fat were removed by blunt dissection, then the peritoneum was incised. The omentum was reflected anterodorsally, exposing the gravid uterus.

The uterus was easily brought to the flank incision, but no portion could be delivered through it. The uterus was incised on its greater curvature, exposing the rump of the fetus. The hindlimbs of the fetus were flexed and delivered, one at a time, then secured with obstetrical chains. Extreme care was required during traction to prevent unnecessary laceration of the uterine incision. The uterus was dry and hair was sloughing from the slightly emphysematous fetus. The fetal membranes were manually separated from the uterus and removed.

The uterine incision was closed with two rows of continuous Lembert sutures. Because of the friability of the peritoneum

Dr. Melby is a general practitioner in Middlebury, Vt.
The author thanks Dr. G. Danks, Ithaca, N.Y., and Dr. A. W. Wright, Vergennes, Vt., for their assistance.

and the large deposits of fat, some difficulty was encountered in closing the parietal peritoneum with a continuous suture of No. 2 plain catgut. The fascia of each abdominal muscle layer was closed with interrupted sutures of No. 2 chromic catgut and it was sprinkled with sterile sulfanilamide. The skin was united with a continuous interlocking stitch of No. 2 nylon and "Smear 62" was applied.

Three million units of aqueous procaine penicillin, 5 Gm. of dihydrostreptomycin sulfate, and 20 thousand units of tetanus antitoxin were injected intramuscularly, and 1 million units of crystalline penicillin was given intravenously.

AFTERCARE AND RESULTS

The total time required for the operation was approximately four hours. The owner was instructed to remain with the patient and keep her in either lateral or sternal recumbency as long as possible. However, the owner found it impossible to prevent the mare's several attempts to rise during the three hours before she was able to stand unassisted.

About 12 hours after surgery, the mare was listless, often stood with the front legs crossed, walked with difficulty, and refused both feed and water. The temperature was 101.7 F., the pulse rate 107, and respiration was accelerated. Four million units of procaine penicillin and 5 Gm. of dihydrostreptomycin in aqueous suspension were injected intramuscularly, and 1,500 units of tetanus antitoxin was given subcutaneously.

On May 11, the general condition of the mare was improved. She would eat small amounts of grass if it was placed in her mouth. A facial paralysis, which persisted for several days, interfered with normal prehension and mastication. The temperature was 103.3 F. and the pulse rate 99, so the treatment of the day before was repeated.

The next morning, the condition of the mare was improved; the temperature was 102.7 F. and pulse rate 94. Five hundred milliliters of 40 per cent dextrose solution was injected intravenously, and the treatment of the previous day was repeated. Later that day, the temperature was 103.0 F. and the pulse rate 85, so 2.5 Gm. of oxytetracycline was injected intravenously.

On May 13, the temperature was 101.7 F., the pulse rate was 85, and the oxytetracycline and dextrose therapy was repeated. On May 14, her condition was unchanged, so 4 million units of procaine penicillin and 5 Gm. of dihydrostreptomycin in aqueous suspension were injected intramuscularly, and 500 ml. of 40 per cent dextrose was given intravenously.

During the next ten days, the mare's temperature and pulse gradually returned to normal and her general condition improved, but injections of penicillin and dihydrostreptomycin were continued. She preferred to have grass brought to her but would graze if necessary. A liberal amount of bran was fed in the grain mixture. She was given a laxative on the seventh postoperative day, but her bowel eliminations did not return to normal until 1 gallon of light mineral oil had been given through a stomach tube.

On the twelfth postoperative day, the skin sutures were removed and, because of a small collection of sanguineous fluid under the skin, a 5-cm. opening was made at the ventral end of the incision to facilitate drainage. Healing was complete in three weeks. Six weeks after surgery, the mare appeared normal in every respect and was ridden daily by the owner.

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Cesarotomy in Two Mares

Cesarotomy was successfully performed in 2 draft mares, in Switzerland, because of the difficulty of delivering their foals which were in transverse presentation and occupying both horns of the uterus. In both cases, the mares were fatigued from labor and the foals were dead so, rather than risk anesthesia, sufficient narcosis was given to halt labor and the operations were performed with the mares standing with their heads held high.

The operation was performed through the flank toward which the foal's head lay. The area was prepared and infiltrated with about 200 cc. of 3 per cent local anesthetic. The vertical incision was started at the level of the lower margin of the tuber coxae extending ventrally 32 to 35 cm. The oblique abdominal muscles were separated

bluntly, as far as possible, then the incision included the border of the rectus abdominis as well as the transversus abdominis and peritoneum. The uterus was turned so the forefeet lay in the abdominal incision, then the uterus was incised with scissors, the forefeet and head grasped, and the foal extracted.

In both cases, extraction was difficult through the small abdominal opening. The uterine hemorrhage, mostly diffuse due to the atonic state of the uterus, was alarming. The larger vessels were ligated and hypophysin was injected through the serosa into the uterine wall, resulting in immediate contraction and control of the hemorrhage.

The uterine incision was closed with a continuous Lembert catgut suture inverting 2 cm. of the edge of the wound. The abdominal incision was closed with a separate line of catgut sutures for each layer of muscle and No. 14 silk sutures in the skin.

In 1 mare, the edges of the skin were merely drawn together with sutures but when these were removed in two weeks, the edges separated and the wound required three to four weeks to heal. In the second mare, the skin was sutured so the edges were everted and primary healing occurred. Antibiotics were applied locally at each stage of the operations and systemically for two or three days until the rectal temperatures were normal.

Both mares developed abdominal edema but both were entirely recovered and able to work in a month.—*W. Graden in Schweiz. Arch. f. Tierheilk. (Jan., 1958): 51.*

Antibody Absorption in Newborn Herbivores.—The permeability of the gut of the newborn herbivore to the protein molecules of milk whey is largely lost within 24 hours after birth. All of the colostrum whey proteins, not only the antibody fraction, are absorbed during this time, but the nonantibody proteins, having a relatively low molecular weight, are rapidly excreted through the kidneys while the antibodies are largely retained. Attempts to maintain the intestinal permeability beyond the first 24 hours were unsuccessful. *—Am. J. Physiol., 191, (1957): 271 (abstr. in Vet. Bull. (May, 1958): Item 1518.)*

A Mandibular Splint for a Dog

WILLIAM P. BARCHFELD, V.M.D.

Apollo, Pennsylvania

In the dog, proper alignment of a fractured mandible can sometimes be a problem, especially if there is oblique, overriding, or multiple fragmentation.

The mandible seems to be relatively resistant to infection. Compound fractures generally heal without complications when properly secured and aligned if the nerve and blood supply are not greatly damaged. Osteomyelitis is rare even when wires which penetrate the bone are left exposed. Reasonable sterility and optimum use of antibiotics are indicated.

Fig. 1—Fixation apparatus for a fracture of the mandible of a dog includes: 2 pair fisherman's pliers; 1 pair curved hemostats; 1 needle holder with scissors; 1 small root elevator (used as drill); 1 formed splint; 1 intramedullary wire; and 1 towel clamp to attach tongue to nose of dog.

Fixation apparatus that can be placed inside the mouth has an advantage in some instances. Such a splint (fig. 1) can be made from an intramedullary pin with a small diameter. A pin 1/16 to 3/32 inch in diameter and from 6 to 10 inches in length and sharpened on both ends will supply all of the support needed. The pin can be bent with pliers and fingers to the desired shape to fit the mouth of the patient.

The device, when styled, serves as a simple spring. The U of the clamp is opened so the sharp, everted ends penetrate, under moderate tension, into the posterior parts of the mandible below and behind the last molar tooth on each side. The U-shaped spring lies on the floor of the mouth in the

Dr. Barchfeld is a general practitioner in Apollo, Pa.

Fig. 2—Dorsolateral view of the head of a Boxer, showing a fracture (arrow) of the right ramus with medial displacement.

Fig. 3 (above)—Dorsoventral view of the head of the same Boxer shown in figure 2, indicating fractures (a, c), with splint secured to the ramus (b).

Fig. 4 (right)—Dorsoventral view of the head of an 8-year-old Pomeranian showing a downward fracture (arrow) of the body of the right mandible.

trough between the tongue and the lingual surfaces of the mandible (fig. 2, 5).

In applying the splint, a shallow hole is drilled into the mandible about 0.5 inch below, and posterior to, the last molar tooth to anchor the ends of the wire. When the splint is in place, small holes are drilled through the mandible in appropriate spots on either side of the fracture to accommodate the stainless steel wire which is passed through these holes and tied around the splint. The incisors and canine teeth are also used for supports for wires which anchor the splint. The wire stays in place when anchored around these teeth because of their opposite angulation. Correct placement and tension of the wire sutures will align and fix the fracture fragments in apposition.

Fig. 5—Dorsoventral view of the head of a Dachshund with a symphyseal fracture (a) and multiple fractures (b) of the right ramus in the area of the canine tooth.

CASE REPORTS

The following illustrations show how the splint was used in 3 dogs.

Case 1.—In a Boxer, 10 months old, a radiograph (fig. 2) showed a fracture and medial displacement of the right ramus, with separation at the symphysis. The splint was applied as described. The right end (fig. 3b) was anchored just anterior to the fracture (fig. 3a). At the symphyseal separation (fig. 3c), a wire was placed around the incisor teeth and the splint. The splint, thus secured at three points, aligned and stabilized the fractured mandible.

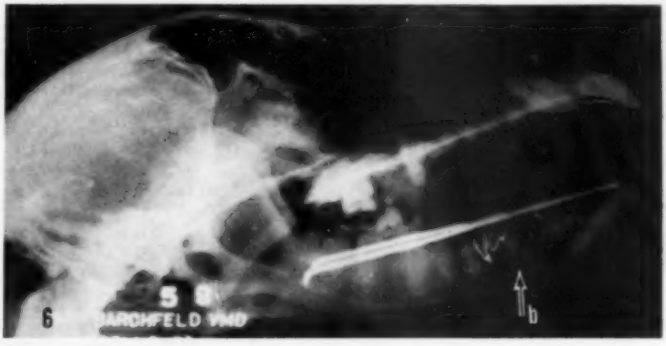

Fig. 6—Lateral view of the head of the same Dachshund shown in figure 5, showing the fracture (b) of the right ramus.

Case 2.—A large Pomeranian, 8 years old, suffered a fracture of the right mandible between the first and second premolar teeth. The ends of the splint were anchored as described (fig. 4). The splint was further secured by wiring it to the mandible on each side. One loop of wire was passed through each fixation wire and around the canine teeth, as well as the incisors.

Case 3.—A Dachshund, 4 years old, had a symphyseal fracture and a compound oblique fracture of the right half of the mandible between the canine and the second premolar teeth. The splint was applied as usual and, in addition, was fastened by

wires around the incisor teeth and premolars on each side (fig. 5, 6).

RESULTS AND CONCLUSION

The use of this splint allows optimum freedom of the jaw. The device is well tolerated and, in our experience, the results have been good.

An Ovine Oddity

J. H. ROSENBERGER

Clinton, Wisconsin

In the spring of 1957, I was called to assist in an ovine dystocia. Upon arrival at the lambing shed, I noticed what appeared to be a thin red ribbon crisscrossed over the straw in the pen. It proved to be a semidried lamb intestine, apparently unraveled by the struggle and movement of the ewe while in labor.

Viscera were protruding from the ewe's vulva. The lamb, except for its viscera, was found entirely within the uterus of the ewe. The semidry lamb was in a ventrotransverse position in the somewhat contracted uterus. The three-quarter Oxford ewe, which had lambed once previously, was still vigorous.

Fig. 1—Partly decomposed lamb showing complete expulsion of the abdominal viscera and an enlarged and cystic liver.

The lamb was delivered after considerable manipulation. Following delivery, the ewe's uterus was found to be ruptured in the dorsolateral portion of the right horn.

Dr. Rosenberger is a general practitioner in Clinton, Wis.

Since the ewe appeared to be in good condition with no excessive hemorrhage, laparatomy and suturing of the torn uterus was suggested but, because of the additional expense, the owner decided against surgery. The lack of hemorrhage indicated that the uterus was probably ruptured at the beginning of labor. As much as possible of the fetal membrane was removed, broad-spectrum uterine tablets were introduced, and some additional supportive treatment was given. Much to everyone's surprise, the ewe made an uncomplicated recovery and was sold that fall.

To substantiate my opinion that this was actually a schistosomus reflexus, several photographs of the specimen were taken and sent to the Ohio State Veterinary College, where this was verified. This anomaly is seen most commonly in dairy cattle and is only rarely observed in sheep and swine.[1]

In the photograph (fig. 1), it appears that the prolapsed viscera were protruding through the pelvic canal of the lamb. Actually, the entire abdominal wall was absent and the spine was flexed and semiankylosed in a manner which apparently thrust the pelvis and hindquarters forward and upward.

This was the first case of schistosomus reflexus I have encountered in sheep and it seems worthy of being reported.

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[1]Roberts, S. J.: Veterinary Obstetrics and Genital Disease. Published by the author, distributed by Edwards Bros. Inc., Ann Arbor, Mich., 1956.

[2]Venske, W. F., Chairman, Department of Veterinary Anatomy, Ohio State University: Personal communication, April 24, 1957.

Torsion of the Uterus in the Mare

Of 52 mares with torsion of the uterus, at a clinic in Germany, 41 (79%) survived following treatment. Of 45 with the cervix open, the torsion was reduced by manual manipulation of the fetus *per vagina* in 34 (75%). Of the 34, 31 (91%) recovered; 2 died of a ruptured uterus.

Of the 11 mares in which the torsion could not be reduced manually, it was reduced by rolling the mare in 10 cases, but 5 of these mares died. At necropsy of the other mare, the uterus was found adherent to the abdominal floor.

Of 25 mares in which the gestation was almost complete, 14 gave birth to live foals.

Of all the mares in which the torsion was reduced by rolling, 50 per cent died. The rolling method seemed to be more dangerous than untwisting the uterus manually.—*F. C. Van der Kaay and C. H. W. de Bois in Berl. u Münch. tierärztl. Wchnschr. (March 15, 1958): 112.*

Evaluation of Semen Quality in the Dog.—Examination of 125 ejaculates of 25 dogs was made to evaluate the semen quality in dogs ejaculated on a moderate regimen and to determine the effects of various frequencies of ejaculation on semen quality, libido, and restoration of semen reserves.

Hand manipulations proved superior for collection, to use of an artificial vagina. With a frequency of one collection for two days, the sperm content remained optimal while more frequent collections reduced the concentration.—*Vet. Bull. (June, 1958): Item 1950.*

Magnesium Sulfate for Euthanasia in Dogs

JOSE B. ARAÑEZ, D.V.M., M.S., and
LUIS B. CADAY, D.V.M.

Quezon City, Philippines

Magnesium sulfate is inexpensive and widely available as a useful drug for euthanatizing dogs. In sufficient volume, a saturated solution given intravenously or intracardially will cause immediate cessation of heart action and respiration. Thus, euthanasia with magnesium sulfate is humane and immediate. It has been recommended that a 1:1 solution of magnesium sulfate dissolved in water by heating will provide a lethal concentration.[1]

This study was made to find the ideal amount and concentration of the solution to euthanatize a dog, using 48 dogs of both sexes, ranging from 9 to 29 lb. in weight, and from 4 to 30 months of age. Three dogs were injected intravenously in the cephalic vein, and 3 intracardially, through the fourth or fifth intercostal space at the level of the costochondral junction, with each concentration of solution.

The 30 dogs given 10, 20, 30, 40, and 50 per cent solutions of magnesium sulfate showed similar antemortem reactions during the injection; the most characteristic were involuntary urination, slight muscular tremors, and an immediate increase in heart beat and respiratory rates, which gradually decreased as death approached. Two of the 30 dogs groaned and 5 involuntarily defecated.

The 18 dogs given 60 or 70 per cent, or saturated, solutions had similar reactions except that the muscular tremors were more intense, resulting in jerking of the limbs. The pupillary changes were variable, some showing constriction at the the start of the injection and dilatation during the second half, while others showed dilation throughout the course of injection, and still others showed dilatation only soon after death. The dosage per pound of body weight used to produce euthanasia in the dogs is shown (table 1).

TABLE 1—Summary of the Range and Average Dose of Various Concentrations of Magnesium Sulfate Solution Required for Euthanasia of Dogs

Concentration	Intravenous		Intracardial	
	Range (cc./lb. body wt.)	Average (cc./lb. body wt.)	Range (cc./lb. body wt.)	Average (cc./lb. body wt.)
10 %	2.17-2.93	2.48	2.02-4.96	3.77
20 %	1.23-1.73	1.53	1.69-2.03	1.89
30 %	1.47-2.40	1.98	1.20-1.55	1.37
40 %	1.20-1.60	1.38	1.23-1.94	1.47
50 %	1.02-1.29	1.14	1.04-1.78	1.31
60 %	1.26-1.36	1.31	0.99-1.09	1.03
70 %	0.45-1.22	0.94	0.63-1.02	0.76
Saturated*	0.62-1.13	0.88	0.60-0.80	0.67

*83 per cent in boiling water.

The required dose of magnesium sulfate varies little whether given intravenously or intracardially, although we found the intracardial injection was more practical and easier to administer than the intravenous injection.

Necropsy of the euthanatized dogs revealed that, with little variation, the hearts were distended and, when incised, the muscles lacked tone; the lungs were congested.

Dr. Arañez is assistant professor, Veterinary Medicine and Surgery, College of Veterinary Medicine, University of the Philippines, Quezon City; Dr. Caday is a recent graduate from the same college.

This is an abstract of an undergraduate thesis presented by the junior author on March 24, 1958, to the faculty of the College of Veterinary Medicine, University of the Philippines, in partial fulfillment of the requirements for the degree of doctor of veterinary medicine. This work was conducted under the direction of the senior author.

The authors acknowledge the help of Dr. V. Taca, city veterinarian of Manila, for providing most of the experimental dogs, and of Dr. A. C. Gonzaga, professor and head, Department of Veterinary Physiology and Pharmacology, from the same college for his suggestions.

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Clinical Data

The Prophylactic Effect of Chlortetracycline, Given Orally, on Bovine Leptospirosis

LEIF M. RINGEN, Ph.D., and WILLIAM OKAZAKI, M.S.

Pullman, Washington

THE CHEMOTHERAPEUTIC effect of antibiotics on leptospirosis has been reported by several workers and will not be reviewed here. Most of these reports were concerned with drugs which are administered parenterally after infection has been established in an animal.

Both chlortetracycline and oxytetracycline have been used as feed supplements in studies on porcine leptospirosis.[1,2] The results of these experiments indicate that these antibiotics, given as oral medication, could eliminate or suppress the carrier condition in most swine.

The following investigation was made to determine the effect of feeding chlortetracycline (Aurofac 10*) as a prophylactic in bovine leptospirosis.

MATERIALS AND METHODS

Twenty calves, 4 to 6 months old, none of which showed evidence of infection prior to their exposure to *Leptospira pomona*, were used—6 were untreated controls and 14 were given Aurofac 10 mixed in their feed, free choice. The quantity given was calculated on the basis of 0.1 mg. (5 calves) and 0.5 mg. (9 calves) of chlortetracycline per pound of body weight per day for 21 days, starting Jan. 28, 1957.

On the eighth day of feeding, these animals, as well as the controls, were exposed to *L. pomona* by placing 3 to 4 drops of culture material on the conjunctiva four times in one day, at two to three hour intervals.

Following exposure, the calves were observed for signs of infection, using fever, rising blood serum antibody titer, and leptospiruria as criteria. For serological studies, the agglutination-lysis test was used. Leptospiruria was determined by dark field examination of urine, together with inoculations of this urine into weanling guinea pigs which were then tested for a rising blood serum antibody titer. The detailed procedure used for studying experimental bovine leptospirosis was described in a previous report.[4]

From the Department of Veterinary Microbiology, College of Veterinary Medicine, State College of Washington, Pullman.

Paper No. 1699, project 1176, Washington Agricultural Experiment Station, State College of Washington, Pullman.

This investigation was supported in part by a grant from American Cyanamid Co., New York, N.Y.

*Aurofac 10 was supplied by American Cyanamid Co., New York, N.Y., and contains 10 Gm. of chlortetracycline per pound of Aurofac 10.

RESULTS

All 6 of the control animals became infected and showed a rising blood serum antibody titer and leptospiruria; and 4 of the 6 showed a febrile response (table 1).

The smaller quantity (0.1 mg./lb. of body wt.) of chlortetracycline had no effect on the prevention of bovine leptospirosis, since all 5 calves became infected and excreted leptospiras in their urine. However, none of the 9 given the antibiotic at the higher level (0.5 mg./lb.) had either a fever or leptospiruria, although 7 of the 9 developed agglutinating antibodies. Their blood serum antibody titers, however, were lower than those of the control animals (table 1).

The 2 calves which were not infected on the first exposure became infected on a subsequent exposure, 74 days after the last day of feeding the antibiotic, and each showed a febrile response, blood serum antibody, and leptospiruria.

DISCUSSION

The prophylactic effect of chlortetracycline was determined by feeding this antibiotic to young calves prior to their exposure to *L. pomona* and continuing this medication past the incubation period. The attempt to simulate field conditions, when exposing the calves, was considered adequate since all of the control animals became infected. If a more severe challenging dose of *L. pomona* had been used, the results might have been different. Also, it is not known how much antibiotic each animal received, or what antibiotic blood serum levels developed.

Prophylaxis apparently was achieved with 0.5 mg. per pound of body weight per day of the antibiotic, but not with 0.1 mg. per pound. Most of the calves fed chlortetracycline at the higher level developed blood serum antibodies, but their titers were much lower than those of the control animals; also, they showed no fever and no leptospiruria.

A good prophylactic should allow the development of antibodies while suppressing

TABLE I—The Prophylactic Effect of Chlortetracycline Feed Medication on Bovine Leptospirosis

Animal No.	Weight (lb.)	Treatment	Febrile response	Serological reponse	Lepto-spiruria
260	203	Chlortetra-	+	± 10^{-2}	+
263	176	cycline:* 0.1	+	+> 10^{-2}	+
264	217	mg./lb. body	+	+> 10^{-2}	+
265	183	wt./day	+	+> 10^{-2}	+
266	150		+	± 10^{-2}	+
206	243	Chlortetra-	−	−†	−
208	202	cycline: 0.5	−	+> 10^{-2}	−
209	273	mg./lb. body	−	+ 10^{-2}	−
211	243	wt./day	−	−†	−
273	295		−	± 10^{-2}	−
275	158		−	± 10^{-2}	−
276	266		−	+ 10^{-2}	−
277	188		−	+ 10^{-2}	−
278	332		−	± 10^{-2}	−
240	—	Controls; no	−	+> 10^{-2}	+
241	—	antibiotic	+	+> 10^{-2}	+
261	176	suppelment	+	± 10^{-2}	+
262	230		+	+> 10^{-2}	+
270	191		+	+> 10^{-2}	+
271	257		−	+> 10^{-2}	+

*Aurofac 10 contains 10 Gm. of chlortetracycline per pound of Aurofac 10; † these animals became infected upon subsequent exposure.

other signs of disease, thus allowing the animal to become immunized. The antibody titers (10^{-2} or higher) obtained in calves fed the higher level of antibiotic were equivalent to, or higher, than those (10^{-1}) obtained in vaccinated animals.[2] It is assumed that animals having titers such as these would be refractory to subsequent infection with *L. pomona*.

The lower level of antibiotic demonstrated no prophylactic effect; all of these calves showed fever, blood serum antibody titers, and leptospiruria. The antibiotic was fed free choice, so some animals probably obtained more than others. However, since there was no evidence of protection in any of the animals, it is assumed that the lack of protection was due to an insufficient quantity of antibiotic in the feed.

The febrile response occurred during the feeding time; therefore, increasing the length of time of feeding probably would not have changed the effectiveness of this level of antibiotic.

SUMMARY

The prophylactic effect of chlortetracycline (Aurofac 10) as a feed medication against bovine leptospirosis was determined by feeding it to calves for one week prior to their exposure to *Leptospira pomona*, and continuing for 14 days post-exposure, on the basis of 0.5 mg. and 0.1 mg. per pound of body weight per day.

None of the 9 calves fed chlortetracycline at the higher level showed a fever or leptospiruria, but 7 developed blood serum antibodies.

All 5 calves fed the antibiotic at the lower level developed fever, blood serum antibodies, and leptospiruria similar to those developed by the 6 control calves after exposure.

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Leptospiral Nephrosis in Calves.—In acute leptospirosis in calves, pathological changes in the kidneys are similar to those in carnivores. After recovery, gray-white foci, representing proliferation of lymphoid cells or connective tissue, remain in the kidneys. In acute cases, necrotic foci are also found in the liver.—*W. Jeleff in Monatsh. f. Vet.-med. (April 15, 1958): 226.*

Leptospirosis has been diagnosed in herds of swine and cattle in every Iowa county this spring. Prophylactic vaccination has been effective but treatment with antibiotics has been unsatisfactory.—*J. B. Herrick in Nat. Hog Farmer (June, 1958): 3.*

Study of the Viability of *Leptospira Pomona* in Frozen Extended Bovine Semen

R. K. JONES, D.V.M., Ph.D.

Lafayette, Indiana

IT HAS BEEN suggested that leptospirosis may be spread among cattle by contaminated semen during natural or artificial insemination. Although such contaminated semen has not been found in nature, the anatomical relationship of the urinary and reproductive systems of the bull make semen contamination possible when leptospiras are discharged in urine. If a bull were a urinary shedder of leptospiras, the disease might be spread in insemination.

This study was made to determine the effect of a process of semen preparation involving glycerolation, antibiotic treatment, and freezing upon the viability of *Leptospira pomona*.

REVIEW OF THE LITERATURE

It was reported that Leptospira survived no longer than 48 hours in Schuffner's medium when frozen in a mechanical refrigerator or carbon dioxide refrigerator.[4] It was also reported that *L. pomona* remained motile less than four hours when held at -20 C.[5] It was found that Leptospira remained motile for seven days when stored at 5 C. in standard semen extender to which penicillin or streptomycin was added, and for one or two days when held at 28 C.[1]

MATERIALS AND METHODS

Leptospira Pomona Cultures.—Two strains were used. One, designated "Johnson," was maintained in Schuffner's medium for several years. The other, designated "Morse,"* had been isolated from swine shortly before the beginning of the experiment. Both strains were grown in Schuffner's medium with 10 per cent rabbit serum for eight days prior to their use as semen extender contaminants.

Aliquots of these were formalized at the time of contamination and the numbers of leptospiras determined, using a Petrov-Hausser chamber and dark field microscopy. The contaminating Morse culture contained 9.6 x 10^8 leptospiras per milliliter, the Johnson culture 1.1 x 10^9 leptospiras/ml. It was estimated that the dosage of leptospiras given guinea pigs was between 35 and 40 million cells. Susceptibility control guinea pigs were inoculated with approximately 1 billion leptospiras.

Preparation of Semen Extender.—The extender was prepared in two portions. Part A was egg yolk citrate buffer to which antibiotics were added in final concentrations of 2,000 μg. of dihydrostreptomycin sulfate, 500 units of penicillin, and 1,000 units of polymyxin B sulfate/ml.

Part B was composed of equal portions of egg yolk buffer and glycerol with a concentration of 500 units of penicillin/ml.

For control purposes, semen extender was also prepared without antibiotics.

Preparation of Contaminated Semen Extender.—One milliliter of Leptospira culture was added to 4 ml. of part A of the semen extender and, in some instances, 1 ml. of freshly collected semen from a bull known to be free of antibody for *L. pomona* also was added. These mixtures were held at 30 C. for 30 minutes, then cooled in a refrigerator at 5 C. At the end of three hours, an additional 9 ml. of part A was added, and 15 ml. of part B was added in five steps during 30 minutes.

Preparation and Storage of Ampules.—After glycerolation, the contaminated semen extender was pipetted in 1-ml. amounts into pull-off ampules and sealed in a Kahlenberg apparatus. The material was then frozen and final storage was in liquid nitrogen at −190 C.

Inoculation of Guinea Pigs.—The frozen material was thawed in ice water just prior to the time guinea pigs were inoculated. The contents of a single ampule was injected intraperitoneally. At the same time, susceptible control guinea pigs were similarly injected with untreated organisms.

Cultural Examinations.—Blood samples were collected by cardiac puncture from each of the guinea pigs on the fifth day after inoculation. Approximately 0.2 ml. of blood was inoculated into 3.3 ml. of Schuffner's medium with 10 per cent rabbit serum in screw-capped vials. Each blood culture was made in triplicate. The inoculated mediums were examined once a week for five weeks. The detection of motile leptospiras in any culture was taken as evidence that leptospiremia had occurred in the guinea pig involved.

Serological Tests.—The agglutination-lysis test was used. The "Johnson" strain of *L. pomona* was used as antigen. Cultures used were 7 to 14 days of age and contained at least 500 leptospiras per low power field. Serum was diluted with saline 1:10, 1:100, 1:1,000, and 1:10,000. Equal quantities (0.1 ml.) of diluted serum and culture were mixed together and held at room temperature

From the Department of Veterinary Science, Purdue University, Lafayette, Ind.

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*Obtained from E. V. Morse, Department of Microbiology and Public Health, Michigan State University, East Lansing.

for at least four hours prior to reading. Serological tests were considered significant if more than 50 per cent of the leptospiras were agglutinated or lysed by a serum diluted 100 times or more.

Serological tests were made on all guinea pigs on the fifth day after inoculation and again on the fourteenth day in trials 1 and 2 and on the twenty-first day in trial 3.

RESULTS

The results are summarized (table 1).

All guinea pig serums tested on the fifth day after the start of each trial were free of *L. pomona* antibody. All uninoculated controls remained free of antibody over the period of observation.

Leptospiremia was detected in guinea pigs inoculated with stock cultures of *L. pomona*, Morse strain, but not in those inoculated with Johnson strain.

Leptospiremia was detected in guinea pigs inoculated with Morse strain contaminated semen extender devoid of antibiotics held at −190 C. for 31 days.

Significant serological response occurred in guinea pigs inoculated with stock cultures of both strains of *L. pomona* and among those inoculated with nonantibiotic treated semen extender contaminated with either strain and held at −190 C. up to 108 days.

Neither leptospiremia nor significant serological response occurred in guinea pigs inoculated with antibiotic-treated semen extender contaminated with either strain and held at −190 C. up to 108 days.

DISCUSSION

It is not surprising that the freezing techniques developed for the preservation of the viability of spermatazoa should also preserve Leptospira. The latter are smaller and possibly less complex forms of life.

Antibiotic treatment of extended glycerolated semen is a common commercial practice, but sensitivity of leptospiral serotypes to antibiotics may be quite variable. It was reported that *L. pomona* was one of the comparatively more streptomycin-resistant serotypes under certain environmental conditions while not under different conditions;[2] also, that motility of the organisms was not necessarily an indication of their ability to propagate.

We found it quite difficult to identify leptospiras frozen in glycerolated extender by direct dark field examination and, consequently, abandoned this as an assay method. The animal-inoculation tests described are more critical and the results more easily interpreted.

The fact that there was a uniformly negative serological response among guinea pigs inoculated with antibiotic-treated material rendered the positive serological findings in those inoculated with nontreated material more significant. Some doubt concerning this conclusion may be supported by the findings[7] that *L. pomona* frozen and thawed in chorioallantoic fluid of hens' eggs stimulated a serological response in calves, although the freezing and

TABLE 1—Results of the Inoculation of Guinea Pigs with Frozen Leptospira Pomona Inocula After Various Treatments

Trial No.	Culture	Inoculum composition	No. of guinea pigs	Evidence of leptospiral infection of guinea pigs	
				Leptospiremia at 5 days	Significant serological response*
1		With semen and antibiotics.	4	0/4	0/4
31 days		w/o** semen with antibiotics.	4	0/4	0/4
after	Johnson	w/o semen w/o antibiotics.	4	0/4	1/4
freezing	culture	Stock culture—not frozen.	4	0/4	4/4
		No inoculum.	4	0/4	0/4
2		With semen and antibiotics.	4	0/4	0/3†
31 days		w/o semen with antibiotics.	4	0/4	0/4
after	Morse	w/o semen w/o antibiotics.	4	2/4	2/4
freezing	culture	Stock culture—not frozen.	4	3/4	3/4
		No inoculum.	4	0/4	0/4
		With semen and antibiotics.	14	0/14	0/12†
3		With semen w/o antibiotics.	3	0/3	1/3
108 days		w/o semen w/o antibiotics.	3	0/3	1/2†
after	Morse	Stock culture—not frozen.	2	2/2	2/2
freezing	culture	No inoculum.	2	0/2	0/1†

*Agglutination-lysis titer of 1:100, or above, at 14 or 21 days after inoculation; **w/o—without (all inocula contained semen extender, with or without semen and antibiotic); †discrepancies in numbers tested versus numbers inoculated accounted for by death loss attributed to collection of first blood sample taken for culture.

thawing process was reported to have "removed infectivity."

One research attempt failed to demonstrate propagability of *Vibrio fetus, Corynebacterium pyogenes, Brucella abortus,* and *Listeria monocytogenes* frozen in antibiotic-treated, extended glycerolated semen, unless the antibiotic was removed from the organisms by washing prior to test inoculation.[3] This was not done in the experiments reported here.

Although the evidence indicates that the antibiotic treatment described will suppress spread of leptospirosis in frozen extended semen, it should not be accepted as the sole means by which the disease is to be controlled in artificial insemination. The threat of the disease is minimized even more by the use of serologically negative sires, the direct treatment of sires which become naturally infected,[6] or the use of a vaccine as a preventive.

SUMMARY

1) The method of preparation and freezing of semen extender described seemed satisfactory for the control of *Leptospira pomona* contamination.

2) *Leptospira pomona* was demonstrated capable of surviving in semen extender not treated with antibiotics at least up to 30 days after freezing and storage at −190 C. On the basis of serological response of guinea pigs, it presumably survives up to 108 days under the same freezing and storage conditions.

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Experimental Ovine Leptospirosis

Both bovine and porcine strains of *Leptospira pomona* produced disease in sheep after subcutaneous inoculation, with leptospiremia occurring on postinoculation days 5 to 8. Meat from these animals may be a potential source of infection for man during this stage. Sheep may contract infection from pigs.

By postinoculation day 8, leptospiras were found in the renal tissues of infected sheep, and leptospiruria started at day 14 and continued for a month, but the organisms were excreted only intermittently and in small numbers. Thus, sheep probably play only a minor role in transmission of the disease.

During five weeks of contact with infected sheep, other healthy sheep, pigs, and goats did not become infected.—*E. V. Morse et al. in J. Infect. Dis., 101, (1957): 129 (Abstr. in Vet. Bull. (May, 1958): Item 1373.)*

Porcine Leptospirosis in Romania.—Agglutination-lysis tests on serum from 1,573 swine at 50 breeding and feeding establishments showed 70 per cent positive to at least one of five Leptospira types, 84 per cent of these reacting to *Leptospira pomona.—Yearbook Inst. Anim. Path., 7 (1957): 77.*

How Diet Zinc May Be Lost

Zinc was removed from solutions containing up to 109 p.p.m. when these were shaken for 1.5 hours with calcium and phosphate supplements. This may account for zinc deficiency in animals whose diet is rich in minerals.—*Vet. Bull. (May, 1958): Item 1529.*

Transmission of Favus of Mice to Man.—A dermatophyte of mice can be acquired by dogs and cats and transmitted to man. Family epidemics due to this organism were found in the Province of Quebec. The name "*Microsporum quinckeanum*" is proposed for the organism.—*Vet. Bull. (May, 1958): Item 1394.*

Lamb Losses Associated with *Clostridium Perfrigens* Type A

BLAINE McGOWAN, D.V.M.; J. E. MOULTON, D.V.M., Ph.D.;
S. E. ROOD, D.V.M.

Davis, and Chico, California

THE PURPOSE of this paper is to present a preliminary report concerning a series of deaths encountered in young nursing lambs, which appeared to be caused by *Clostridium perfringens* type A. The clinical, necropsy, histopathological, and bacteriological findings seemed identical to those of a disease of mature sheep which apparently has been described[4] only in Australia.

The losses investigated occurred on six ranches in California during the spring of 1958; on five within a 13-day period, and on one some four weeks later. Of the 2,400 lambs involved, approximately 4 per cent died.

The affected lambs were thrifty and ranged in size from 20 to 70 lb. Deaths occurred in uncastrated, nondocked ram lambs, wether lambs, and ewe lambs. In 4 cases, the lambs and their mothers were on native pasture and, in 2 cases, on improved pasture. Five ranches were located in the Sacramento Valley and one in the Coast Range. Rainfall was above normal during this period and, therefore, green feed was abundant although watery.

CLINICAL MANIFESTATIONS

Some lambs were found dead by the owners, while others lived six to 12 hours after illness developed. Depression and lassitude were the first signs noticed, and these were particularly evident when the lambs were forced to move any distance. Some lambs collapsed while being moved.

On examination, pale, icteric conjunctivas and scleras were observed, as well as increased respiration and marked hemoglobinuria. The temperatures ranged from normal to 106 F. Weakness and exaggerated respiration increased until death, which usually occurred without a struggle.

With the exception of 1 purebred lamb which was given blood transfusions, death terminated all naturally occurring cases in which icterus and hemoglobinuria developed.

NECROPSY FINDINGS

Necropsies on 7 lambs with field cases all showed generalized icterus and pale mucous membranes. The small intestine was diffusely reddened in 1 lamb and peritoneal petechiae were found in another. In all 7, the livers were enlarged, friable, pale, and slightly yellowish; in 2, there were also disseminated white foci. None of the 7 showed liver infarction. The spleens were normal.

The kidneys were swollen, soft, and reddish brown. Dark red stippling was observed on the surface of all kidneys, and cone-shaped, reddish brown areas resembling infarcts were observed in three. In all 7 lambs, the urinary bladders were distended with clear, dark red urine, and petechiae of the mucosal surface were observed in 2.

The lungs of 3 lambs were dark red, moist, and doughy, and the tracheas contained pink froth. Epicardial and endocardial petechiae were present in 2 lambs, and the mandibular and pharyngeal lymph nodes of 2 were enlarged, reddened, and juicy.

Histopathology.—In the livers of all 7 lambs there was a slight increase of mononuclear cells in the portal areas. Many hepatic lobules exhibited zonal necrosis which was segmental or central. Necrotic areas often formed broad anastomosing bands that, in some cases, involved entire lobules. Necrotic cells showed karyorrhexis, karyolysis, and increased acidophilia of cytoplasm.

In more advanced cases, polymorphonuclear leukocytes had invaded necrotic hepatic tissue. The livers did not contain excess hemosiderin. Kupffer cells were not involved, and there was no excess bile pigment in canaliculi or bile ducts.

In the spleens, there was a slight increase of red blood cells in the red pulp, but no excess of hemosiderin or of reticular cells, and no lymphocytic hyperplasia.

In the kidneys of all 7 lambs, there were degenerative changes in the proximal and, to a lesser extent, in the distal convoluted tubules. Secondary interstitial invasion by mononuclear cells had occurred in 3 animals. The outstanding renal change

Drs. McGowan and Moulton are engaged in teaching and research at the School of Veterinary Medicine, University of California, Davis; Dr. Rood is a general practitioner in Chico, Calif.

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was the presence of hemoglobin casts (fig. 1), which stained orange-pink with hematoxylin and eosin and were found in convoluted and collecting tubules. The dark red conical areas, seen grossly, consisted of groups of tubules greatly distended by

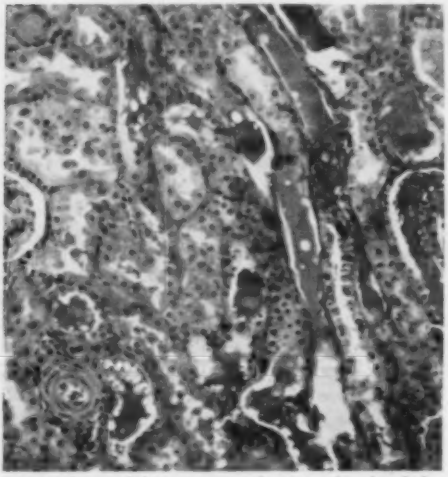

Fig. 1—Hemoglobin casts in the convoluted tubules of a kidney from a lamb with enterotoxemia type A.

hemoglobin casts. Hemosiderin granules stained the epithelium of convoluted tubules in 1 animal. The glomeruli were normal. The kidneys of 5 lambs were stained by the Levaditi method for leptospiras, and all showed negative results.

The lung changes consisted of pulmonary hyperemia and edema. No abnormalities were found in the central nervous system. The lymph nodes, which were grossly abnormal in 2 lambs, were hyperemic and edematous microscopically.

BACTERIOLOGICAL AND EXPERIMENTAL FINDINGS

When direct smear of the intestinal contents was made, the predominant organism was a medium sized gram-positive bacillus. Isolation was accomplished by first inoculating one loopful of gut contents, from each of four areas of small intestine, into cooked-meat medium and incubating at 37 C. for 24 hours, under anaerobic conditions. These cultures were then streaked on tryptose blood agar and incubated under nitrogen for 48 hours.

Individual colonies that demonstrated *beta* hemolysis surrounded by a zone of incomplete hemolysis were selected and inoculated into cooked-meat medium. This medium seemed to support adequate toxin production for our purposes.

Seitz filtrates of intestinal contents directly from 1 lamb with a naturally occurring case and broth culture filtrates from 2 lambs with field cases produced hemoglobinuria and death in mice, when given intravenously whereas, after these filtrates were neutralized for 30 minutes with *Cl. perfringens* type A antitoxin, they were innocuous for mice.

Procedures for determining the presence of *Cl. perfringens* lecithinase were conducted as follows:[4]

One milliliter of test fluid was incubated for one hour at 37 C., with 0.5 ml. of indicator. Human serum was used for this purpose. Opalescence appeared within 24 hours, indicating the presence of a lecithinase.

Mixtures were then made containing 1 ml. of test fluid and 1 ml. of 1:10 dilution of *Cl. perfringens* type A antitoxin. After 30 minutes at room temperature, 0.5 ml. of indicator was added, and the tubes were incubated for one hour at 37 C. The results were read 24 hours later, and the inhibition of opalescence by the type A antitoxin indicated the presence of *Cl. perfringens alpha* or *theta* toxin, both of which are produced by *Cl. perfringens* type A.[5]

Three lambs, ranging in weight from 15 to 25 lb., were inoculated intravenously with culture filtrates. The first lamb was given 22 ml. of broth filtrate which produced a pale icteric sclera and transient hemoglobinuria, followed by recovery in 24 hours. The second and third lambs received 30 and 40 ml. of culture filtrate, respectively.

Immediately after inoculation, the lambs showed increased respiration and uneasiness followed by lassitude and, 45 minutes after inoculation, hemoglobinuria was observed in both lambs. The lamb given 40 ml. was dead in two hours, which was too soon for typical lesions to develop. The lamb given 30 ml. became progressively weaker, showed a pale icteric sclera in three hours, and died 17 hours after inoculation. The lesions in this lamb were identical to those described in the lambs with the naturally occurring cases.

TABLE 1—Hemoglobin and Packed Cell Volume Values in 1 Lamb Inoculated Intravenously with Clostridium Perfringens Type A Culture Filtrate

Hours after inoculation	0	1½	6	13
Hemoglobin (Gm./100 cc. of blood)	12.2	8.8	6.8	3.8
Packed cell volume (mm.)	42.0	24.0	8.0	1.5

Hemoglobin and packed cell volume determinations (table 1) were made on the second lamb. These data agree quite closely with the hemoglobin determinations and erythrocyte counts reported after intravenous inoculation of sheep with purified culture filtrate of *Cl. perfringens* type A.[1]

Copper determinations on liver (75 mg./10 Gm.) and kidney tissue (105

mg./10 Gm.), using the method described by the Association of Official Agriculture Chemists, were considered normal.

Dark field examinations of urine from 2 lambs with field cases and hamster inoculations were negative for Leptospira.

DISCUSSION AND SUMMARY

The data presented concerning the clinical, necropsy, and histopathological findings closely resembled those of the disease "enterotoxemic jaundice of sheep" as described in 1936 in Australia.[6] However, the Australian report concerned adult sheep only, whereas the California deaths were confined to suckling lambs. It is felt, nevertheless, that this age difference is not sufficient evidence to attempt to establish two separate diseases.

Since 1936, the role of *Clostridium perfringens* types B and D in producing animal diseases has been considerably clarified. In recent years, *Cl. perfringens* type C has been found to produce a disease entity in sheep[4] and cattle,[3] and now *Cl. perfringens* type A is also incriminated in diseases in sheep. Because it is becoming increasingly more common to refer to two of these diseases as enterotoxemia type D and enterotoxemia type C, the authors suggest that the disease described be termed enterotoxemia type A.

The evidence presented here to establish *Cl. perfringens* type A as the causal organism in a series of deaths in suckling lambs is believed sufficiently strong to establish a new disease entity of sheep in California and the United States.

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Lipodystrophy in a Black Leopard

A. C. HERZBERGER, D.V.M.

Colorado Springs, Colorado

The purpose in writing up this case history is not to help other practitioners with their black leopard practice, but simply to offer them something different. Many surprising and unusual cases have been seen in my experience with zoo animals, but none more so than when I opened the abdomen of a leopard at the Cheyenne Mountain Zoological Gardens near Colorado Springs, Colo.

CASE REPORT

On Feb. 19, 1957, I was called to look at an adult male, black leopard which had been refusing part of his food for sometime and had now quit eating. Preliminary examination disclosed nothing except a normally bad-tempered animal with a bloated abdomen and a much duller coat than the other animals.

It took several of us about an hour and a half to bring the leopard under control. His temperature was normal, and an enema brought no results. After careful palpation of the abdomen, a diagnosis of abdominal ascites was made. Because of the leopard's fighting disposition and the noisy conditions, auscultation of the thorax was not possible. The animal was released.

Permission was granted to do a laparotomy on this valuable animal, since there might be a possibility of surgical correction of the cause of the ascites.

The following day, the leopard was caught, anesthetized with pentothal, and prepared for aseptic surgery.* A midline, ventral incision into the peritoneal cavity resulted in the gushing forth of nearly 4 gallons of a white, milklike liquid containing numerous pieces of a rubbery, free-floating, pale gray tissue. On careful examination of the completely drained abdomen, no tumor formation or other condition that might stop the flow of lymph through the lymphatic system was found. The lymph nodes appeared normal. The only noticeable change was a slight swelling of the liver.

Dr. Herzberger is a general practitioner in Colorado Springs, Colo.

*The author acknowledges the assistance and advice of Dr. T. R. Knowles.

It was then decided to examine the thoracic cavity for tumor formation or other involvement of the thoracic duct. Therefore, tracheal intubation was performed and a resuscitator attached. Upon incising the left side of the diaphram, there again was a flow of chyle-like fluid, this time from the pleural into the peritoneal cavity. When an examination of the left half of the thoracic cavity revealed no cause of the effusion, the resuscitation was stopped and death followed in a short time.

On necropsy, nothing more than already mentioned was found, particularly nothing that might have occluded the lymphatic ducts.

HISTOPATHOLOGICAL FINDINGS†

The lymph nodes had a normal follicular architecture, with no evidence of tumor involvement but with a large amount of infiltration of foamy phagocytes, particularly into the periphery of the nodes. The nodal sinusoids were markedly dilated and frequently contained clusters of pigmented phagocytes.

The fibro-fatty tissue surrounding the nodes also contained large clusters of pigmented phagocytes, as well as collections of inspissated fibrinous material in which large numbers of leukocytes and large amounts of basophilic cellular debris were apparent. Some of this fatty tissue showed focal zones of necrosis with accumulations of fibrinous material, some of which showed hyaline transformation. In these areas, there were collections of polymorphonuclear leukocytes, pigmented phagocytes, and foamy phagocytes, as well as numerous lymphocytes.

The lymphatic channels were markedly dilated and some were stuffed with foamy phagocytes. Others contained thrombi composed principally of red blood cells, fibrinous material, many lymphocytes, and some pigmented phagocytes.

The liver had a normal architecture but with marked congestion in some places. The capsule showed some prominently swollen cells and, occasionally, within it and on it there were collections of fibrinoid material with basophilic cellular debris, large numbers of foamy phagocytes, and large numbers of leukocytes. There was no evidence of inspissation of bile within the bile capillaries. Within the liver were several small zones of necrosis containing collections of polymorphonuclear leukocytes and fibrinous material and some golden brown staining material, but no evidence of specific infecting reagents or organisms.

The rubbery pale gray tissue found in the peritoneal cavity was an inspissated fibrinous material composed of droplets of fatty material, red blood cells, numerous lymphocytes, and some polymorphonuclear leukocytes and large numbers of degenerated mononucleated phagocytes.

†By Dr. R. W. Utich, St. Francis Hospital.

DISCUSSION AND CONCLUSIONS

The principal causes of chylous effusion in both the pleural and peritoneal cavities are tumors, granulomatous infections, and trauma. Since tumors and granulomatous inflammation had been almost completely eliminated, and since there was no known trauma, it would seem probable that this was a case of intestinal lipodystrophy, similar to Whipple's disease in man.

The etiology of such a disease is unknown, but it has been suggested that it may be a bile salt metabolism defect. It is believed that the chylous ascites is a late development in the disease, due to blockade of the lymphatic channels with fatty material and thromboses.

Spontaneous Chylopneumothorax in Man

Fluoroscopy of a 25-year-old man, who had experienced shortness of breath for one week and dyspnea for 24 hours, revealed a fluid level at the fourth intercostal space on the right side. Thoracentesis yielded 1,400 cc. of milky fluid after which dyspnea decreased. Radiographs showed air and fluid in both pleural spaces and both lungs partially collapsed.

Thoracotomy revealed that the pulmonary pleura was replaced by a thin layer of tissue which puckered the lung in areas and provided many minute air leaks, the source of the pneumothorax. Chyle appeared to be oozing from many points over the mediastinal parietal surfaces, but there was no disruption of the thoracic duct.

The diagnosis was malignant mesothelial cell growth. The patient died and, at necropsy, the tumor was found to extend through the diaphragm to the liver and to the right adrenal gland and kidney.

No report of a similar, spontaneous, bilateral chylopneumothorax or of other cases of chylous fluid associated with a pleural mesothelioma was found in the literature. *—J.Am.M.A. (April 12, 1958): 1909.*

An Intradermal Test for Turkey Ornithosis.—An intradermal test for the detection of psittacosis exposure in turkeys is described in *Am. J. Hyg. (66,* 1957: 245). The serological and skin tests agreed in 70 to 85 per cent of the infected birds studied. *—Vet. Bull. (May, 1958): Item 1448.*

Erysipelas in a Flock of Chickens

C. L. VICKERS, D.V.M., and BERT W. BIERER, V.M.D.

Columbia, South Carolina

INFECTION WITH *Erysipelothrix rhusiopathiae*, while not common in chickens, was reported as early as 1905.[2] It was first reported in the United States in 1943[1] and, since then, at least 2 other cases have been recorded.[1] It is the purpose of this paper to report the first confirmed case of erysipelas in chickens in South Carolina.

HISTORY

On Jan. 18, 1958, 7 white leghorn hens, about 11 months old, were brought to the Clemson College diagnostic laboratory so the cause of their illness could be determined.

There were 3,000 hens in the flock, all housed in one building. The building was divided into three sections by means of wire partitions, with approximately 1,000 hens in each section. For about six weeks, the hens had been dying at the rate of 2 to 6 a day. Since there was a history of leukosis in the flock, the owner at first assumed this to be the cause of death. Most of the losses occurred in one section of the house, although there were some deaths in all three sections.

CLINICAL SIGNS AND PATHOLOGICAL FINDINGS

The sick birds had a yellowish diarrhea, ruffled feathers, inappetence, and tended to isolate themselves. Some were found dead in the nest, while others were noticeably sick for four or five days before death. Cannibalism was present in the flock. Egg production had dropped from 60 per cent to about 38 per cent.

At necropsy, enlargement of the livers and spleens and enteritis was noticed in all the birds. Necrotic areas, from pinpoint size to about one fourth of an entire lobe, were present in the livers of 2 birds, lesions of visceral lymphomatosis were found in 2, and hyperemia of the cloacal mucosa was found in 3. The ovaries appeared active, but at least 5 birds had ruptured ova. The unruptured ova were semisolid in consistency, and the yolk material was thickened, having almost a cooked appearance. The ova were slightly shrunken and somewhat distorted.

BACTERIOLOGICAL FINDINGS

An organism, tentatively identified as *Erysipelothrix rhusiopathiae*, was isolated by cultural methods from the spleens, livers, and hearts of several of the birds. While this organism is often isolated from swine and turkeys in this laboratory, confirmation of this finding was obtained at another laboratory,* which identified the organism as *Erysipelothrix rhusiopathiae*.

While awaiting confirmation, a separate saline suspension of both a known Erysipelothrix culture and the unconfirmed culture was made. The known culture had previously been recovered from a turkey. Four 1-year-old hens were injected with these saline suspensions; hen 1 was injected intramuscularly with 0.5 cc. of the unconfirmed culture; hen 2 was similarly injected with the known culture; hen 3 was injected subconjunctivally in the lower right eyelid with 0.5 cc. of the unconfirmed suspension; and hen 4 was similarly injected with the known suspension.

DISCUSSION AND RESULTS

Within 24 hours following the injections, the injected eyelids of hens 3 and 4 were slightly swollen and, 48 hours after injection, there was a marked erythematous swelling in both hens, involving the fleshy surface of the entire right side of the face, completely closing the right eye.

Hen 3 died 60 hours after injection. At necropsy the only lesions were an enlarged liver and spleen. Erysipelothrix was recovered from the spleen, liver, and heart. The swelling on hen 4 gradually subsided, and within a week she appeared completely recovered. Hens 1 and 2 were never visibly ill.

These hens were kept in wire cages suspended from the ceiling. Several hens kept

From the Diagnostic Laboratory, Clemson College Livestock Sanitary Department, Columbia, S. Car.

The authors thank Dr. Richard D. Shuman, Bacterial and Mycotic Disease Section, Agricultural Research Service, U.S.D.A., Beltsville, Md., for his assistance; also Mr. W. A. Dennis, Jr., of Scattered Farms Feed, Cameron, S. Car.

*Bacterial and Mycotic Disease Section, Animal Disease and Parasite Division, Agricultural Research Service, U.S.D.A., Beltsville, Md.

in cages immediately adjoining these never showed signs of illness. However, a hen allowed to run free underneath the cages the second and third days after the injections was found dead the third day after being returned to a cage, and Erysipelothrix was recovered from the internal organs.

Following the tentative diagnosis of erysipelas, the owner was advised to add penicillin to the feed of the entire flock and to isolate all the sick birds and treat them by individual intramuscular injections of penicillin. He decided to isolate the sick birds and treat them. Acutely ill birds were given 300,000 units of aqueous penicillin intramuscularly, and the less severely affected received 150,000 units.

There was a marked improvement in the treated birds within 48 hours and, within 5 days, losses had stopped. Within two weeks, egg production increased to about 61 per cent.

SUMMARY

Erysipelas in a flock of chickens is described. Signs of the infection were inappetence, a yellowish diarrhea, and a drop in egg production. On necropsy of 7 affected birds, enlarged livers, spleens, and ruptured ova were found. The unruptured ova were semisolid, with the yolk being thickened, having an almost cooked appearance.

Hens experimentally injected in the inner surface of the eyelid with a saline suspension of the isolated *Erysipelothrix rhusiopathiae* became ill, while those injected intramuscularly were not visibly affected.

One hen exposed to droppings from the experimentally infected birds died, while several hens in wire cages adjoining the infected birds never became ill.

Isolation and individual treatment of the sick birds, with penicillin by intramuscular injection, resulted in recovery of the flock.

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Fluoridated Water and Chick Growth.—When chicks were given water containing up to 100 p.p.m. of sodium fluoride for six to ten weeks, it had no significant effect on growth, water consumption, and bone ash content.—*Vet. Bull. (May, 1958): Item 1507.*

Pesticide Poisoning of Livestock

The newer chemical insecticides and herbicides are a boon to agriculture, but they can poison animals, when eaten, and sometimes from surface contact. The chlorinated hydrocarbon insecticides such as DDT, TDE, methoxychlor, benzene hexachloride, isodrin, and endrin affect primarily the central nervous system, resulting in severe depression or convulsions and death by respiratory paralysis. Animals not fatally poisoned usually recover completely if given a quieting agent, good nursing, and uncontaminated feed.

The organic phosphorus compounds such as parathion, malathion, dipterex, chlorothion, guthion, and others are closely allied to the nerve gasses developed for chemical warfare, and act primarily by adversely affecting the animal's nervous system. For these, atropine sulfate is a good antidote but must be given in large doses, part of it intravenously for quick action. The degree of intoxication depends on the size of the agent particles in the emulsions as well as upon the amount ingested or contacted.

Herbicides rarely poison animals, because of the unpalatability of the treated plants.—*Agric. Res. (May, 1958): 5.*

Organophosphate Pesticide Destroyed in the Rumen.—Parathion and three other related compounds were rapidly destroyed when 100 p.p.m. were added to bovine rumen fluid. This may explain the apparent lack of toxicity of these agents for cattle.—*Vet. Bull. (May, 1958): Item 1477.*

Toxicity of Antibiotics in Veterinary Medicine.—Toxic and allergic reactions to antibiotics are of no practical importance in veterinary medicine and, in contrast with human medicine, resistance of bacteria is of little importance.

Of 341 pathogenic strains of staphylococci from mastitis in cows, 4 were resistant to penicillin, 21 to streptomycin, and none to chlortetracycline. Of 128 coliform strains, four were resistant to streptomycin and one to chlortetracycline.—*H. Keller in Schweiz. Arch. f. Tierheilk. (Nov., 1957): 651.*

Effectiveness and Toxicity of Sustained Action Quinacrine Hydrochloride in Dogs

FREDERICK SPERLING, Ph.D.

Arlington, Virginia

THE PROPERTY of quinacrine as a teniafuge has been known since 1940.[1] Its effect is probably due to direct irritation causing detachment, rather than to any lethal action on the tapeworm.[2] Link and Smith[3] reported successful use of quinacrine as a teniafuge in dogs. During necropsy, after treatment, they found occasional small tapeworms in the gut. It was their opinion that these tapeworms represented regeneration from undetached scolices. Following along the line of this latter report, a study was conducted to investigate the toxicity and effectiveness of a sustained action formulation of quinacrine hydrochloride (Keybrin*).

MATERIALS AND METHODS

Ten mongrel pups were allotted to two groups. Group A consisted of 4 males and 2 females, ranging in weight from 2.3 to 6.0 kg. Group B consisted of 2 males and 2 females, ranging in weight from 3.7 to 5.1 kg. Proglottids or ova, in concentrated or flotation specimens of the stools or in thick fecal smears, were demonstrated in all 10 dogs before treatment.

The dogs were fed a dry dog food twice daily and water was given ad libitum. Records were kept of the daily food consumption and weekly body weights. Each animal was housed individually in a run in air-conditioned quarters maintained at 72 to 75 F., with relative humidity ranging from 40 to 70 per cent. All animals were given intact tablets of quinacrine hydrochloride (QH) after being fasted overnight. The fast was continued for four hours after giving the tablets. The tablets were stored in brown bottles in a closed cabinet in order to avoid photoreaction.

Group A.—Of the 6 dogs, 5 were given a single tablet (100 mg.) of sustained action QH per 3.6 kg. of body weight, or fraction thereof, following overnight fast. One was given a second treatment two weeks after the first.

Fecal examinations were then made twice weekly for one month. Hematological determinations were made on blood obtained from an ear of each dog just before treatment and at the end of the one month of observation. This included erythrocyte counts, total and differential leukocyte counts, and hemoglobin determinations with a Spencer Hb meter. After one month, each dog was euthanatized for necropsy. The liver, kidneys, and spleen were carefully examined grossly, and the gastrointestinal tract, from the esophagus to the cecum, was removed, slit open, and examined for parasites and pathological changes with a hand lens.

Group B.—These 4 dogs were given an initial dose of QH (100 mg.) and, starting two weeks later, were given similar doses weekly for five consecutive weeks. Fecal examinations were made twice weekly. One week after the last dose, the dogs were euthanatized for necropsy. Hematological

TABLE I—Increases in Body Weights of 10 Dogs, Following Treatment with Quinacrine Hydrochloride

Animal		Weeks observed	Dose		Initial body wt. (kg.)	Final body wt. (kg.)	Gain in body wt. (%)†
No.	Sex		No. tablets	Total mg.			
1	M	4	2	200	6.0	8.0	33.3
2	F	4	2*	200	2.3	3.6	56.5
3	M	4	2	200	4.7	6.2	31.9
4	F	4	2	200	4.0	6.8	70.0
5	M	4	2	200	4.4	6.9	56.8
6	M	4	2	200	3.8	6.8	79.0
7	F	7	12**	1,200	4.8	7.1	48.0
8	M	7	12**	1,200	3.8	6.0	57.9
9	M	7	12**	1,200	4.4	7.4	68.2
10	F	7	12**	1,200	3.7	5.9	59.5

*Second tablet given after two-week interval; **2 tablets per week for six weeks; †average weight gain (%) for group A was 54.6 in four weeks and 58.4 for group B in seven weeks.

Dr. Sperling is director, Sperling Laboratories, Arlington, Va.

*Keybrin is a product of Key Corp., Miami, Fla.

TABLE 2—Blood Cell Counts in 10 Dogs Just Before Treatment with Quinacrine Hydrochloride and Just Prior to Necropsy

Animal No.	Dose: No. tablets	Dose: Total mg.	Erythrocytes (10^6 cmm.)	Leukocytes (10^3 cmm.)	Differential count§: N	E	B	L	M	Hb (Gm./100 cc. blood)
1	2	200	(1†)5.68	21.60	53	0	3	30	14	10.5
			(2‡)5.94	29.60	57	3	3	36	1	10.6
2	2*	200	(1)5.01	15.90	67	0	2	26	5	11.0
			(2)4.97	20.70	28	2	3	65	2	11.7
3	2	200	(1)5.47	16.10	62	0	3	29	4	11.0
			(2)5.42	17.30	59	4	2	33	2	10.4
4	2	200	(1)4.30	23.35	63	0	2	26	9	10.0
			(2)4.69	23.55	34	1	1	59	5	11.3
5	2	200	(1)4.79	21.85	64	0	2	30	4	12.8
			(2)5.03	18.95	46	1	3	47	3	13.0
6	2	200	(1)5.11	21.00	51	0	1	45	3	13.0
			(2)5.90	16.30	32	0	0	65	3	13.0
7	12**	1,200	(1)5.92	25.10	32	4	0	60	4	10.5
			(2)4.76	25.00	15	0	0	79	6	11.0
8	12**	1,200	(1)4.34	17.50	22	2	0	71	5	9.0
			(2)5.62	10.90	32	1	0	60	7	11.5
9	12**	1,200	(1)4.49	21.95	51	3	2	42	2	10.0
			(2)5.10	16.90	33	2	0	61	4	11.5
10	12**	1,200	(1)5.85	14.65	48	6	3	35	8	10.0
			(2)5.80	11.90	40	2	0	53	5	12.0

*Second tablet given after two-week interval; **2 tablets per week for six weeks; † 1 = prior to treatment; ‡ 2 = prior to necropsy; § N = neutrophils, E = eosinophils, B = basophils, L = lymphocytes, M = monocytes.

determinations were made just prior to the first dose and to necropsy.

RESULTS

During the entire testing and observation period, all animals, with 1 exception, ate well and all gained weight (table 1). Vomiting and anorexia were absent.

The blood cell counts of both groups of dogs are shown (table 2). There were no indications of the development of blood dyscrasias. At necropsy, the liver and kidneys of all dogs were normal. One dog in group B had an enlarged spleen (0.8% of body wt. compared with the normal 0.4-0.5%). This was probably due to some cause other than dyscrasia.

Some pathological changes were found in the gastrointestinal tract of some animals, especially those of group A. Since necropsies were performed four weeks after the treatment in 5 of these dogs, and two weeks after treatment in the other, the treatment could not have been responsible for the lesions. In 4 of the 6 dogs, many scattered, elliptical, somewhat raised hyperemic areas measuring about 0.5 by 3.0 cm. were found in the jejunum. These resembled Peyer's patches.

The stomach walls of dog 2, the dog with the low food intake, were hyperemic. It had been given a second dose of 100 mg. of the drug, two weeks after the first, because of persistent signs of tapeworms. However, tapeworm segments continued to appear in the stools and, at necropsy, large masses of tapeworms, 50 to 100, of both Taenia sp. and Dipylidium sp. were recovered from

TABLE 3—Worms Recovered at Necropsy from the Gastrointestinal Tracts of 10 Dogs Treated with Quinacrine Hydrochloride

Animal No.	Dose: No. tablets	Dose: Total mg.	Necropsy weeks after last dose	Worms recovered
1	2	200	5	42 roundworms
2	2*	200	5	17 roundworms 50-100 tapeworms
3	2	200	5	14 roundworms
4	2	200	5	14 roundworms 1 tapeworm
5	2	200	5	6 roundworms
6	2	200	5	9 roundworms
7	12**	1,200	1	1 roundworm 6 hookworms
8	12**	1,200	1	3 roundworms
9	12**	1,200	1	1 roundworm 7 hookworms
10	12**	1,200	1	1 roundworm 1 tapeworm

*Second tablet given after two-week interval; **2 tablets per week for six weeks.

the jejunum. Obviously, this small dose was unable to cope with this heavy infection, which probably was responsible for the gastritis and low food intake. No gastroenteritis was found in any of the other dogs.

In 2 dogs, 1 in each group, a single small tapeworm, Dipylidium sp., was found at necropsy. These undoubtedly represented either regeneration from an undetached scolex or reinfection, since fleas had not been removed from the animals. These instances were similar to those previously reported.[2]

The numbers and types of worms recovered from the small intestine at necropsy are shown (table 3). All of the worms were found in the jejunum, some as high as the duodenojejunal junction, but none in the ileum. Except for the 1 dog, all evidences of tapeworm infection disappeared after treatment.

CONCLUSIONS

1) As demonstrated by necropsy, tapeworms were eliminated in young mongrel dogs by sustained action quinacrine hydrochloride (Keybrin) in 7 of 10 proved cases of tapeworm infection.

2) The drug, given in amounts up to 200 mg. per week for six weeks, produced no evidences of toxicosis or undesirable side reactions in young mongrel dogs weighing 3.7 to 4.8 kg. at the beginning of treatment.

3) Tapeworms were effectively eliminated by treatment with as little as 200 mg. of the drug, given in a single dose to dogs weighing 3.8 to 6.0 kg.

4) A single dose of 100 mg., given to a dog weighing 2.3 kg., was ineffective in an instance of an extremely heavy infection with tapeworms.

5) Two instances of small tapeworms found at necropsy probably represented reinfection or regeneration from a remaining undetached scolex.

6) Sustained action quinacrine hydrochloride appears to be an effective teniafuge.

References

[1]Culbertson, J. T.: The Elimination of the Tapeworm *Hymenolepis Fraterna* from Mice by the Administration of Atabrine. J. Pharm. Exptl. Therap., *70*, (1940): 309-314.

[2]Sheraf, A., and Shikate, I. M.: *In Vitro* Studies on the Anthelmintic Effect of Some Antimalarial Drugs. Am. J. Vet. Res., *16*, (1955): 15-17.

[3]Link, R. P., and Smith, J. C.: Treatment of Canine Tapeworm with Atabrine. J.A.V.M.A., *125*, (1954): 461-462.

A Hemorrhagic Disease of Dogs

An infectious, fatal, hemorrhagic disease, probably of virus origin, occurred in dogs in Romania. At first, only 3- to 4-month-old pups were affected but later it occurred in older dogs.

There was fever, anorexia, apathy, hemorrhages from mucous membranes, petechia in the skin, hematuria, bloody diarrhea, thrombocytopenia, and prolonged clotting time, with death in one to two days in acute cases or in two to three weeks in subacute cases. Most of these dogs were immune to distemper.—*Vet. Bull. (May, 1958): 1433.*

Complement-Fixation Test for Rabies

Antigen for the complement-fixation test was prepared by triple freezing of a 16.6 per cent saline suspension of the brain under test. The antibody was serum from hyperimmunized rabbits. In 103 tests, the results agreed closely and were obtained more rapidly than with animal inoculation. The test was of particular value when Negri bodies were not found in the brain. —*Vet. Bull. (May, 1958): Item 1415.*

Control of Rabies Along the Mexican Border.—The reduction of numbers of wolves, coyotes, and foxes in a zone 150 km. (93 miles), on both sides of the United States-Mexican border, has greatly reduced the incidence of rabies in wild animals in the area.—*Vet. Bull. (May, 1958): Item 1416.*

Toxoplasmosis in Cats

Toxoplasma were recovered, by the inoculation of mice with brain tissue, from 34 of 140 cats in Tennessee, and from 4 of 35 cats in South Carolina.

Serums from 100 of the 175 cats were positive to the dye test and almost all cats with titers of 1:256 or more were found to be infected.—*Vet. Bull. (May, 1958): Item 1410.*

Carbohydrates for Young Pigs.—Newborn pigs can utilize only lactose and glucose but, after ten days, they can assimilate sucrose, maltose, and dextrin.—*Vet. Bull. (May, 1958): Item 1517.*

The Effect of Pelletizing on Vitamins A and E

BERT W. BIERER, V.M.D., and
C. L. VICKERS, D.V.M.

Columbia, South Carolina

In making a routine assay of feed samples in connection with an eye disease problem in turkeys, it was found that a pelletized feed was significantly below the vitamin A and vitamin E claim of the feed manufacturer. The principal source of the vitamin A in the feed was a synthetic vitamin A preserved by a special wax coating. The principal source of the vitamin E was d-*alpha*-tocopherol acetate, a synthetic product reported by its manufacturer to be quite stable.

In view of the fact that pelletizing of feeds is accomplished with the utilization of steam and pressure, it appears to be a logical deduction that a partial destruction of certain vitamins may result.

Materials and Methods

Two feed samples were obtained, one nonpelletized and the other in the form of pellets. The samples were obtained from fresh shipments of turkey breeder ration, prepared by the same mill. The feed manufacturer's claim was that the feeds were identical with the exception that one batch had been pelletized.

The samples were obtained from bags opened for the purpose and, when shipped for assay, consisted of approximately 1 lb. of each feed in separate cardboard containers.

TABLE I—Effect of Pelletizing on Vitamins A and E in Turkey Breeder Ration

Ration	Vitamin A (units/lb.)	Vitamin E (units/lb.)
Pellets	4,950	30.0
Nonpellets	7,140	29.9

Both samples were shipped by air express to the Wisconsin Alumni Research Foundation (Madison, Wis.) with the request that each sample be assayed for vitamins A and E content.

From the office of the Clemson College Livestock Sanitary Department, Columbia, S. Car.

Prepared in cooperation with the American Association of Veterinary Nutritionists.

Results and Conclusions

The assay report on each feed sample (table 1) supports the following conclusions:

1) Evidence is presented that pelletizing results in a significant destruction of vitamin A. This loss may be approximately 32 per cent.

2) Pelletizing does not appear to have any effect on the vitamin E content.

3) It appears that a feed formulated along the lines of minimum recommendations may become a vitamin A deficient feed when pelletized.

Oils, Penicillin, and Bloating

The effect of oil as a prophylactic agent against bloat in 25 steers (av. 620 lb.) on alfalfa pasture was tested, at Iowa State College, in 1956. The steers were allotted to two groups, with the treated and control groups alternating every two weeks. The water-dispersible oil (a lard derivative), when added to the drinking water (1 or 2%), greatly reduced the incidence and severity of bloat in the treated steers.

In the many cases of severe bloat which occurred, an attempt was first made to remove the gas via stomach tube. As this usually failed, 100 ml. of oil was then injected directly into the rumen. In 12 of 13 cases, this resulted in a release of gas from the ingesta in 15 to 40 minutes, with free gas then being eructated or removed by stomach tube. The steer which died was moribund when the oil was injected.

At the end of the pasture season, when the remaining steers were confined to a feedlot and fed alfalfa hay with a grain mixture, there was less bloating but, again, the frequency and intensity of the bloat was less while the steers were given oil-treated water to drink. The oil seemed palatable since the treated animals consistently drank more water.

Bloating was markedly increased when young, lush alfalfa was fed, but it declined as the quality of the alfalfa deteriorated. Bloating was intense during a 16-day period while the alfalfa was growing rapidly after late summer rains; during four days of the 16, all of the steers showed some degree of bloat.

In a second experiment with 15 calves (520 lb.), the treated animals were each

given 75 mg. of procaine penicillin daily via gelatin capsule. Bloating was somewhat less severe in the animals while on treatment but the difference was less than when oil was used. Penicillin did not offer sufficient protection to justify its use. It seemed to be effective initially but not after it had been used for several days or weeks, whereas a level of ¼ lb. of oil per animal per day greatly reduced bloat.—*J. Anim. Sci (May, 1958): 374.*

Effect of Ingested Paraffins on Ruminants

Single doses of 200 ml. of heavy or light liquid paraffin of medicinal quality did not affect the appetites of cows. However, a single dose of 200 ml. of odorless kerosene or a light grade of paraffin of high isoparaffinic content temporarily reduced feed intake 40 to 90 per cent, requiring three to four days for the cows to recover. The anorexia occurred much quicker when this oil was introduced directly into the omasum, indicating that the effect was postruminal.

When 75 ml. of the heavy medicinal liquid paraffin was given twice daily for 26 days, it did not affect the yield of milk or the fat and solids-not-fat content of the milk, but it caused a continuous fall in the carotene and vitamin A content of the blood with a parallel fall in the carotene and vitamin A levels in the butterfat. (Recovery required about three weeks.) This fall was probably due to an interference by the oil with the absorption of carotene from the intestine. Tocopherol absorption was also reduced but not xanthophyll absorption. The butter made from this product had a lighter color than butter from control cows. The effect on carotene and vitamin A levels was proportional to the paraffin oil dosage level.—*Vet. Bull. (April, 1958): Items 1156 through 1161.*

[Editor's note: The above information verifies what some practitioners have long recognized, *i.e.*, that paraffin oils, even in small quantities, are remarkably effective in most cases of bloat. The latter item indicates that a good thing should not be overdone.]

Effect of Gibberellic Acid in Broiler Rations.—Since gibberellic acid stimulates the growth of numerous plants, some of which might be used as poultry feed, the effect of feeding such plants was tested on 160 chicks. These plants had no significant effect on the growth, morbidity, or development of the treated birds.—*Poult. Sci. (March, 1958): 490.*

Protein Malnutrition in the Pig

Pigs fed a diet containing only 4.5 to 6.5 per cent of a protein, after 13 days of age, failed to grow, showed signs of severe protein deficiency after three to four months, and died after four to six months. Death occurred much later when the low-protein diet was started after the pigs were older. —*Vet. Bull. (May, 1958): Item 1519.*

Effect of Milk Components on Calf Diarrhea.—In a study on the influence of various components of milk-type diets on the consistency of feces of calves, milk fat (3%) was the only factor investigated which significantly reduced the laxative effect of the diet. Increasing the mineral content to four times normal produced a moderate diarrhea under some conditions, whereas increasing the mineral to ten times normal under other conditions did not. Increasing lactose to twice normal resulted in a slight diarrhea. Prevention of curd formation, with sodium citrate, did not increase the incidence of diarrhea.—*J. Dai. Sci. (May, 1958): 662.*

Toxicity of Nitrates and Nitrites for Dairy Cattle.—Nitrate poisoning was suspected when 51 cattle died, within 48 hours of the first signs of illness, on two dairy farms in Puerto Rico. Both had been fed high-nitrogen forage.

Therefore, in four trials involving 14 dairy cattle, the nitrate content of the ration was gradually increased to 1.3 to 1.7 per cent potassium nitrate content on a dry weight basis and this ration was fed for 14 to 60 days. All animals continued to gain weight and none showed signs of poisoning. Since the forage fed to the two herds which suffered losses was not more than 1 per cent potassium nitrate on a dry weight basis, it was concluded that deaths were not due to nitrates in the forage.—*J. Dai. Sci. (May, 1958): 694.*

Editorial

Frustrating Terminology: Panleukopenia or What?

An abstract in the *Veterinary Bulletin* (May 1958, item 1434) reports a suggestion that the name "viral agranulocytosis" be used for the viral disease of cats which has been called by many names, including "panleukopenia."

Naming a disease appropriately is not a simple task. Not only are there no set rules but there is often a period—months to years—of uncertainty regarding the basic character of a disease which makes choice of a satisfactory designation difficult. For example, what was once called "contagious abortion" in cattle, a name which indicated only one sign of the disease, was next changed to "Bang's disease," named after an investigator, and finally to "bovine brucellosis" when the causal organism was properly classified.

It may be practicable to use names of men or geographical areas to designate a disease temporarily while investigations are being completed, but permanent names should be based on the clinical or pathological characteristics, the etiological factors involved, or a combination of these.

Terms such as "plague," "distemper," "pest," and "cholera," are too indefinite to be satisfactory, and frequently lead to confusion when translated to other languages. How many Americans know what disease is meant when "fowl plague," or "fowl pest," are referred to? Foreign veterinarians are probably equally confused when "fowl cholera" is mentioned. Hog cholera, a disease which originated and was named in this country, unfortunately is called "swine fever" in many other lands. More confusing is the fact that the term "cholera" is applied to a vibrioid epidemic of man, a viral epizootic of swine, and a Pasteurella epornitic of chickens.

The above terms, which indicate a general illness, are holdovers from an era when little was known about microorganisms. However, they seem to have "squatters' rights" which will not readily be relinquished. This illustrates the urgency of an early establishment of proper names for newly recognized diseases.

The Feline Disease

Perhaps most confusing is this viral disease of cats, which, over the past seven decades, has been known by a frustrating assortment of names, including "feline distemper," "cat fever," "show fever," "cat plague," "yellow vomit," "gastroenteritis," "collibacillosis," "infectious feline enteritis," "panleukopenia," and "agranulocytosis." A new name was used almost every time the disease was investigated. Some of these names indicate the total effect of the disease while others are based only on prominent pathological characteristics. Etiological factors apparently were slighted when names were considered.

Because of its peracute and destructive nature, this disease is difficult to name. It is characterized by marked depression, severe dehydration, drastic hypoplasia of the blood-forming organs and rapid disappearance of the cellular elements of the blood (leukocytes may drop from 16,000 to 500 in 12 hours and, in case of survival, may again increase to the original level in 24 hours). In addition, there is necrosis of the epithelium of the small intestine—especially the ileum, proliferation of the reticulo-endothelial tissues, edema and hemorrhage of the lymph nodes, acute hyperthermia, formation of intranuclear inclusions at certain phases of the disease and, with recovery, there is hyperplasia of the bone marrow.

In 1939, at Rochester University and at Harvard University, medical scientists, while studying anemia in man, found that in this acute disease of laboratory cats almost all the leukocytes and a proportional number of erythrocytes disappeared from the peripheral blood. They found that intranuclear inclusions developed first in the reticulo-endothelial cells in the mesenteric lymphoid tissue and, later, in the crypts of the glands of the terminal ileum.

Because of the many pathological changes in this disease, it would seem unwise to limit the name to "viral agranulocytosis" and thus ignore the many other lesions and remarkable characteristics. Of the names currently used, "feline distemper" would seem the most appropriate were it not that this confuses the disease with canine distemper.

If a satisfactory name for this feline disease can not be devised, there should at least be agreement on one of the most satisfactory past or present names so that the present confusion can be terminated.

ABSTRACTS

Development of the Heart in Bovine Embryos

This investigation was based on 6 serially sectioned cow embryos (*Bos taurus*), recovered 21 and 22 days after insemination. The developmental stages of bovine embryonic hearts correspond closely to the stages described for human embryos of similar age and somite number.

Following the fusion of the paired endocardial tubes to form a simple tubular structure at the six-somite stage, the heart rapidly develops dilations and sulci which indicate the future bulbar, ventricular, and atrial regions. Within the period of the nine- and ten-somite stage, the paired halves of the bulbus and ventricle fuse, and the dorsal mesocardium gradually disappears. A true ventral mesocardium is lacking entirely. A characteristic bending of the cardiac tube, beginning in the ventricular region, of ten- and 12-somite embryos produces a U-shaped bulboventricular loop. As the atria fuse and come to lie dorsal to this loop, the heart assumes a spiraled S shape. By the 20-somite stage, the sinus venosus is distinguishable, and the heart is capable of maintaining some circulation throughout the yolk sac, central nervous system, liver primordium, and allantoic diverticulum.

Some aspects of the cardiac jelly were discussed, in relation to the primary contractions of the myocardium.—[*Marlene R. Grimes, J. S. Greenstein, and R. C. Foley: Observations on the Early Development of the Heart in Bovine Embryos with Six to Twenty Paired Somites. Am. J. Vet. Res., 19, (July, 1958): 591-599.*]

Assessment of Bovine Liver Function

The mean fractional clearance in January for one group of normal, mature, nonlactating dairy cows, between five and 20 minutes after the injection of 1 Gm. of sulfobromophthalein, was 0.230 ± 0.043 $min.^{-1}$ with a mean half-time for dye disappearance of 3.11 ± 0.64 minutes.

In May, another group of mature, nonlactating dairy cows showed fractional clearances of 0.203 ± 0.025 $min.^{-1}$ Decreased fractional clearance was observed in animals with clinical cases of diffuse fascioliasis, multiple liver abscesses, and disseminated embolic suppurative hepatitis.—[*C. E. Cornelius, G. S. Theilen, and E. A. Rhode: Quantitative Assessment of Bovine Liver Function, Using the Sulfobromophthalein Sodium Clearance Technique. Am. J. Vet. Res., 19, (July, 1958): 560-566.*]

Elimination of Phenothiazine by Dairy Cows

The feces, urine, and milk from two groups of cows (milking about 10 lb./day) treated with phenothiazine were analyzed for elimination products of the anthelmintic. In one group of 5 cows, each given 100 Gm. of phenothiazine N.F. (boluses), the average total recovery of the drug was 81.5 per cent (62.4 Gm. in the feces, 19.0 Gm. in the urine, and 0.075 Gm. in the milk). The drug persisted after administration for an average of 79.0 hours in the feces, 112.0 hours in the urine, and 71.0 hours in the milk, with peak eliminations averaging 30.0, 28.4, and 27.6 hours, respectively. Fat-free milk samples became slightly pink on standing under refrigeration at the time of peak elimination.

For comparison, 2 cows were each given 100 Gm. of micronized phenothiazine powder (capsules). The average recovery totaled 77.3 per cent (22.7 Gm. in the feces, 54.3 Gm. in the urine, and 0.288 Gm. in the milk). Elimination times after drug administration averaged 71.5 hours for feces, 132.5 hours for urine, 86.5 hours for milk, with average peaks at 33.0, 26.0, and 26.3 hours, respectively. Fat-free milk samples became decidedly pink on standing under refrigeration.

The primary elimination product in milk has been identified as phenothiazine (probably conjugated in some manner).

Tests on pooled milk samples from 8 dairy cattle given 2 Gm. of phenothiazine N.F. per day indicated that the elimination of phenothiazine in milk was less than 2 p.p.m. These milk samples soured readily and did not become colored under any circumstances.—[*T. Richardson and A. C. Todd: Elimination of Phenothiazine by Lactating Dairy Cows. Am. J. Vet. Res., 19, (July, 1958): 610-619.*]

Pulmonary Adenomatosis in Iowa Cattle

Pulmonary adenomatosis in cattle is an acute, noninfectious, respiratory disease characterized by sudden onset and short duration, varying degrees of pulmonary emphysema and edema, and hypertrophy and hyperplasia of the respiratory epithelium. This last tissue change gives rise to the term "adenomatosis," due to the tumor-like proliferation of cells lining a glandlike cavity (alveolus) in the stroma of the lungs.

Some aspects of pulmonary adenomatosis in cattle have been found to be similar to Montana progressive pneumonia, jagziekte, and maedi in sheep; to epizootic adenomatosis in the Icelandic sheep; to jagziekte-like disease in the horse; to pulmonary emphysema in cattle; and to "silo-filler's disease" in man.

Clinical observations, etiological studies, case histories, and the macroscopic and microscopic findings were presented.

The answer to the question of the pathogenesis of this disease may revolve around the alterable chemical constituents of food plants and the appropriate bacterial flora necessary for their digestion.—[*Vaughn A. Seaton: Pulmonary Adenomatosis in Iowa Cattle. Am. J. Vet. Res., 19, (July, 1958): 600-609.*]

Effect of Milk on Trichostrongylid Nematodes

Significantly fewer *Haemonchus placei* were recovered at necropsy from experimentally infected calves fed milk alone than from calves fed grain and hay in addition to milk. Also, fewer *H. placei*

were recovered from unweaned calves than from weaned calves, whether experimentally or naturally infected. In natural infections, unweaned calves were found to have fewer Cooperia spp. and *Oesophagostomum radiatum*, but approximately the same number of *Ostertagia ostertagi* as weaned calves after 54 days on pasture. Milk in the ration of the host significantly reduced the size of *H. placei;* however, spicular measurements remained within the normal range reported for this species.

Significantly smaller numbers of *Trichostrongylus axei* and *Trichostrongylus colubriformis* were recovered from unweaned rabbits than from their weaned litter mates, 18 days after experimental infection. However, when 4 calves were experimentally infected with *T. axei, T. colubriformis,* and *O. ostertagi,* no difference in worm numbers could be found between the weaned and unweaned pairs.—[*George H. Rohrbacher, Jr., Dale A. Porter, and Harry Herlich: The Effect of Milk in the Diet of Calves and Rabbits upon the Development of Trichostrongylid Nematodes. Am. J. Vet. Res., 19, (July, 1958): 625-631.*]

Fungi in the Crop of the Turkey

Candida albicans and *Saccharomyces tellustris* occurred normally in the crops of turkeys. The microflora growth in the crop of the turkey was affected by the feed eaten by the bird. Turkeys fed a ration containing adequate amounts of all vitamins, salt mixtures, soybean oil, soybean protein, and cerelose as the source of carbohydrates had many more bacteria and fungi than turkeys fed the same ration with starch substituted for the cerelose.

The *C. albicans* and *S. tellustris* isolated from the crops of these turkeys did not ferment in a 3 per cent solution of cerelose or starch. However, fermentation did occur in a 3 per cent concentration of cerelose in a solution of Difco purple base broth, but not in a similar solution with starch replacing the cerelose. These observations suggest that specific nutrients are essential for the growth and multiplication of *C. albicans* and *S. tellustris* and that they are absent in a solution of only cerelose and starch. The utilization of certain vitamins by the fungi in the crop may result in a decrease of vitamins available for the host.—[*Anthony S. Manfre, H. O. Wheeler, G. L. Feldman, R. H. Rigdon, T. M. Ferguson, and J. R. Couch: Fungi in the Crop of the Turkey. Am. J. Vet. Res., 19, (July, 1958): 689-695.*]

Pendulous Crop in the Turkey

Pendulous crops were produced experimentally in young turkeys fed a ration containing 52.9 per cent cerelose. These crops regressed in size and apparently became normal either when the birds were changed to a commercial ration or when starch was substituted for cerelose as the source of carbohydrate.

Saccharomyces tellustris, a gas-producing fungus, grows and multiplies rapidly *in vitro* in the presence of cerelose. This fungus occurs in large numbers and produces much gas in the crops of turkeys fed the cerelose ration. The constant internal pressure stretches the muscles in the wall of the crop, resulting in the loss of muscle tone which is characteristic of this type of pendulous crop.—[*R. H. Rigdon, T. M. Ferguson, G. L. Feldman, H. O. Wheeler, and J. R. Couch: A Study of the Mechanism of the Experimentally Induced Pendulous Crop in the Turkey. Am. J. Vet. Res., 19, (July, 1958): 681-688.*]

BOOKS AND REPORTS

The Conquest of Bovine Tuberculosis in the United States

As stated in the foreword by Dr. J. Arthur Myers, "only one person in the world could have written this story," namely, the author, Howard R. Smith, better known as "H. R." to the hundreds with whom he worked over the years on livestock conservation and bovine tuberculosis eradication. This is a story of people, of events, of organizations which played such an important part in one of the most ambitious disease control programs of our times.

It tells of the public health as well as the economic aspects of bovine tuberculosis; the methods developed for its systematic and wide-scale diagnosis; the efforts to obtain national, state, and county legislation for cooperative testing programs and indemnification of owners for reactors. It tells also of opposition to the program and how it was overcome. Much of the real interest in the booklet's story is its chronicle of incidents and personalities, sidelights which could be written only by one who devoted himself so whole-heartedly to livestock loss prevention in general and effective control of a bovine plague in particular.

The booklet should be in every library on livestock disease.—[*The Conquest of Bovine Tuberculosis in the United States. By Howard R. Smith. 32 page booklet; paper cover. Published by the author, Somerset, Mich., 1958. Price $1.00, single copy, postpaid; 75 cents each in quantity.*] — J. G. HARDENBERGH.

The Guinea Pig in Research

As an experimental animal, the guinea pig has been used to study infectious diseases, allergies, and scurvy. This report on the nutritional requirements of this animal should make possible studies of metabolic disorders common to it and to human beings.

The author also discusses the average blood values, blood pressure, pH values of the digestive organs, and the value of the guinea pig in sensitization, radiation, and pharmacological research.—[*The Guinea Pig in Research. By Mary Elizabeth Reid. 87 pages; paper bound; 7 halftone plates; 12 tables; extensive bibliography. Human Factors Research Bureau, Inc., National Press Building, Washington 4, D. C., 1958. Price not given.*]

THE NEWS

Colonel Trum, V.C. (Ret.), U.S. Army, Appointed to Harvard Medical School Post

Lieutenant Colonel Bernard F. Trum (COR '35), who retired from the Army Veterinary Corps on June 30, 1958, has accepted an appointment in the department of pathology of Harvard Medical School. His principal assignment is to plan, build, and operate an animal facility for the receiving, care, and research of animals other than small rodents.

Colonel Bernard F. Trum

Colonel Trum, a native of Natick, Mass., received his preveterinary training at Boston College and his D.V.M. degree from Cornell University in 1935. He entered the Veterinary Corps the same year and received special training at the Army Medical Center, the University of Kentucky, and the Massachusetts Institute of Technology.

During World War II he served, among other assignments, as chief of the Veterinary Section, Office of the Surgeon General, Advance Section Communication Zone, ETO, and was awarded five battle stars and a Bronze Star. In 1949-1950, he was professor of Zootechnics at the University of San Simon in Bolivia.

From 1951 to 1956, Colonel Trum was professor of Zootechnics at the University of Tennessee. He was then appointed to the staff of the University of Tennessee-AEC agricultural research program at Oak Ridge and served as military director of veterinary radiological health courses at the Institute of Nuclear Studies there from 1953 to 1956. Since then, he has been research coordinator, Army liaison officer, and veterinarian in the Division of Biology and Medicine, AEC, Washington.

He is the author and co-author of nearly 40 scientific publications in the fields of equine diseases, radiobiology, and related subjects, and has been a frequent contributor to the programs of veterinary medical associations and other scientific societies.

Colonel John Rust to Head Life Science Exhibit in Geneva, Switzerland

Colonel John H. Rust, U. S. Army Veterinary Corps, and chief of the Veterinary Pathology Section of the Armed Forces Institute of Pathology, has been selected as chairman for life science exhibits by the Atomic Energy Commission and the Department of State, at the United Nations Organization's 2nd International Conference on Peaceful Uses of Atomic Energy. The Conference will be held in Geneva, Switzerland, Sept. 1-13, 1958.

Purpose of the conference is to promote an exchange of scientific information on atomic energy between member nations. Many scientific papers will be presented and in addition films, books, and exhibits are being prepared especially for the conference delegates.

Colonel Rust, as chairman of the life science exhibits, has the special responsibility of determining the theme and the subjects to be exhibited in the areas of agriculture, biology, and medicine. He is being assisted by Dr. Lloyd J. Roth, chairman of the Department of Pharmacology, Medical School, University of Chicago.

Exhibits will stress low power reactors and use of short lived isotopes in life science research. The labeling of substances of biological interest with carbon-14 and tritium will be demonstrated and it will show some of the ways in which it is accomplished chemically by growing plants in special "greenhouses." Also to be portrayed will be some of the recently developed methods by which tracers have been effectively used to advance learning in the life sciences. Many of the exhibits will be operating laboratories where visiting scientists can be shown by actual demonstration, often by the scientists who developed the technique, how to solve the special or unique problems existing in their own countries.

The Geneva exhibit and conference will be one of great interest to the world's scientific community. The scientific papers presented will fill many volumes and constitute an important reservoir of scientific knowledge for future investigators.

Selection of Colonel Rust as chairman of this exhibit recognizes the increasingly important role which veterinary medicine and the profession are taking in science today.

Dr. Willard Wright Retires—Will Continue as Consultant

Dr. Willard H. Wright (GW '17), head of the Laboratory of Tropical Medicine at the National Institutes of Health, retired on June 30, 1958, after nearly 40 years of government research work. He began his career of government service in 1919 as a veterinary inspector with the then Bureau of Animal Industry. From 1925 to 1928, he was with the Maryland State Board of Agriculture and subsequently returned to the BAI where he was associate parasitologist in the zoological division until 1936.

Dr. Wright then was appointed senior zoologist in the National Institutes of Health, U.S. Public Health Service, was acting chief of the zoological division in 1938-1939 and served as chief from 1939 to 1947. In the latter year, he was made chief of the Laboratory of Tropical Diseases. In addition to his degree in veterinary medicine, he obtained the M.S. degree from American University in 1931 and the Ph.D. degree in parasitology in 1935. He was also assistant director of the National Microbiological Institute for a number of years.

Dr. Wright held various other appointments and lectureships and was recognized as one of the nation's leaders in the field of tropical disease research. He will continue as a consultant to the Public Health Service, attending international medical conferences in Stockholm and Lisbon this fall.

Eleventh World Health Assembly Achievements

The Eleventh World Health Assembly concluded its three-week session in Minneapolis on June 13, 1958, under the chairmanship of Dr. Leroy E. Burney, Surgeon General, U.S.P.H.S.

Among its highlights and activities were: a stepping up of malaria eradication programs; greater emphasis on research programs to which the United States has contributed a special fund of $300,000; a comprehensive program to deal with health implications in peaceful uses of atomic energy; and advances toward a global small pox eradication program.

The Assembly was attended by delegates from all the 85 active member states. The next session will be held at its headquarters in Geneva, Switzerland, in 1959.

Animal Care Meeting

The ninth annual meeting of the Animal Care Panel will attract more than 500 scientists, veterinarians, and animal technicians from the United States and from several foreign countries to the Hamilton Hotel in Chicago, Dec. 3, 1958, to begin a three-day discussion on the care of laboratory animals.

Thirty-one reports by scientists engaged in medical and biological research on methods to improve conditions under which their experimental animals are housed, fed, treated, and used will be presented.

Subjects will range from "Nutrition of Rabbits" to "Laboratory Animals in Cardiovascular Research." Of particular interest will be a report by Mrs. A. S. Monroney, president of WARDS (Welfare of Animals Used for Research in Drugs and Surgery), a newly formed, volunteer group of citizens and doctors interested in promoting high standards of care for laboratory animals. Last year, WARDS raised $20,000 for the expansion and modernization of the animal quarters at Georgetown University, College of Medicine and Dentistry, in Washington, D.C.

The second day of the meeting will be devoted to a symposium on animal quarter design and construction. The officers of the Animal Care Panel are: Dr. Jules S. Cass, Kettering Laboratory, University of Cincinnati, School of Medicine, president; Dr. Bennett J. Cohen, director of the U.C.L.A. Medical Center Vivarium, vice-president; Dr. R. J. Flynn, Argonne National Laboratory, Lemont, Ill., secretary-treasurer.

Consultant Group to Poultry and Egg Industries Formed

Announcement was received of the formation of a group of consultants to the poultry and egg industries on July 1, 1958, known as Cliff D. Carpenter Associates.

Headed by Cliff D. Carpenter, D.V.M., for 14 years president and executive officer of the Institute of American Poultry Industries in Chicago, the consultant group is made up of authorities in various phases of poultry and egg production, processing and marketing. The group will specialize in governmental relations, plant operations, analyses, sanitation, and public relations.

Dr. Carpenter has been associated with the poultry industry ever since he graduated from New York State Veterinary College at Cornell in 1920, first in diagnostic laboratory work, then in specialized poultry practice, in California.

During World War II, he served with the U. S. Department of Agriculture and mapped the nation's wartime poultry and egg production; he became special assistant to the director of the War Food Administration and organized the National Poultry Advisory Council. He also helped develop special wartime veterinary programs concerned with nutrition and postwar planning.

The new consulting group will be located at 1207 Emerald Bay, Laguna Beach, Calif.

STATE BOARD EXAMINATIONS

BRITISH COLUMBIA—November, 1958 (usually end of the second week; Vancouver. G. L. Stovell, British Columbia Veterinary Association, 3187 West 43rd Ave., Vancouver 13, secretary.

AMONG THE STATES AND PROVINCES

Illinois

Women's Auxiliary.—The quarterly meeting of the Women's Auxiliary to the Central Illinois V.M.A. was held in conjunction with a clinic sponsored by the Central Illinois V.M.A., at the Nicholas Hotel, Springfield, in June, 1958.

Mrs. A. Van Walleghen, president, presided over the meeting and Mrs. Duke Morland read the minutes and the treasurer's report. Mrs. A. L. Starkey, McLean, president of the Auxiliary to the Illinois State V.M.A., addressed the meeting. Mrs. T. M. Wise, Effingham, was the auxiliary's guest.

In the evening, the auxiliary members joined their husbands for a cocktail hour and dinner. Dr. Thomas M. Wise, president, Illinois State V.M.A., presented an interesting after-dinner speech.

The officers serving the auxiliary this year are: Mrs. A. Van Walleghen, Jerseyville, president; Mrs. Donald J. McDermith, Nokomis, vice-president; and Mrs. Paul B. Doby, Springfield, secretary-treasurer.

s/MARY ANN WALLEGHEN, *President*

Kansas

Women's Auxiliary.—The Women's Auxiliary to the Kansas V.M.A. held its semiannual meeting in connection with the twentieth annual conference of the Kansas V.M.A. at Manhattan, on June 5-7, 1958.

Mrs. Dean S. Folse, Manhattan, program chairman for the brunch, arranged a half hour of folklore music with a commentary by Dr. Earl Davis, head of the Department of English at Kansas State College. Mrs. George Eberhart, Manhattan, conducted this portion of the meeting which was held in the Terrace Room of the Wareham Hotel. She extended a welcome from the wives of the veterinary school's faculty to the auxiliary.

Mrs. Leonard Mohney, of Pratt, auxiliary president, presided at the business session.

The following members were appointed to serve on the nominating committee: Mrs. George Chapman, Beloit, chairman; Mrs. Merle Henrikson, Emporia; Mrs. C. W. Bower, Topeka; and Mrs. L. W. Pottroff, Liberal.

A report of the constitution revision committee appointed in February, 1957, resulted in the revised document being submitted for its first reading. This constitution will be presented for the second reading and adoption at the annual meeting of the auxiliary to be held in Wichita in January, 1959.

s/MRS. K. MAYNARD CURTS, *Secretary.*

Michigan

Dr. Mootz, Jr., Named Inspector in Charge of the Meat Inspection Station in Detroit.—Dr. C. E. Mootz, Jr. (MSC '44), has been named inspector in charge of the Detroit meat inspection station of the U.S.D.A. He succeeds Dr. M. L. Cravens who retired on June 30, 1958.

Dr. Mootz, who took over his duties June 16, 1958, was formerly at the Chicago station where he served as supervisor.

Minnesota

Graduates Feted with a Dinner at Minnesota University.—The graduating seniors of the College of Veterinary Medicine were given a "recognition dinner" by the faculty of the college on June 13, 1958.

Dean W. T. S. Thorp extended a cordial welcome and introduced the distinguished guests. Dr. George Hartle, Minneapolis, president of the Minnesota State Veterinary Medical Society, congratulated the students on their attainment and then administered the veterinarian's oath.

Dr. George Hartle, president, Minnesota State Veterinary Medical Association, addressing the seniors at the faculty-senior dinner held at the University of Minnesota in June. Dean W. T. S. Thorp (seated) presided at the dinner.

The principal address of the evening was presented by Dr. Wayne O. Kester, Brigadier-General (Ret.), V.C., U.S.A.F., and past-president of the AVMA. His speech, entitled "What Is Ahead for the Veterinary Profession," sought to remind the neophyte veterinarians that "school is never out for the successful veterinarian." General Kester emphasized the importance of supporting and taking part in local, regional, state, and national societies and associations, stating that whatever a veterinarian does to strengthen himself or herself will also serve to strengthen the veterinary profession as a whole.

Missouri

Kansas City Association.—The June meeting of the Kansas City V.M.A. was held in the Aztec Room of the Hotel President on June 19, 1958.

During the business meeting, the subject of ethical telephone listings was thoroughly discussed. The association's program included: Dr. Glen L. Dunlap—Pregnancy Test Demonstration; Dr. H. C. Ashby—California Mastitis Test Demonstration; Dr. L. D. Jernigan—X Ray Diagnosis—Dwarfism in Cattle; Dr. F. E. Coley—Poisoning Diagnosis; Dr. E. J. Krautmann—Traumatic Gastritis Diagnosis; and Dr. Heinz Goldman—Blood Urea, Blood Sedimentation, and Bacterial Sensitivity Tests.

s/FRANK O'DONNELL, *Secretary.*

New York

New York Association.—The Veterinary Medical Association of New York City, Inc., held its first family picnic at the Anthony Wayne Recreation Area, about five miles south of Bear Mountain, N. Y., June 22, 1958.

The association hopes to make this family picnic an annual affair.

s/NATHAN Z. HOWARD, *Chairman.*

Ohio

Ohio State V.M.A. Presents Dr. Merrick with Public Relations Service Award.—Dr. Byron P. Merrick (OSU '23), Berlin Heights, Ohio, is the recipient of the 1957 Public Relations Service Award of the Ohio State V.M.A.

The presentation, made during a one-day symposium in June, 1958, on the latest developments in veterinary medicine, was held at Sisson Hall, the new veterinary science building at Ohio State University. The award, an engraved, bronze replica of the State of Ohio, was presented by Dr. Joseph A. McCoy, Washington Court House, president of the Ohio State V.M.A.

Dr. Byron P. Merrick (right) is pictured receiving the Ohio State V.M.A.'s Public Relations Service Award from Dr. Joseph A. McCoy.

Dr. Merrick, one of four brothers to be graduated from the College of Veterinary Medicine at Ohio State, was cited for "his outstanding contributions in furthering the public relations of the veterinary profession in the State of Ohio." Dr. Merrick is a member of Alpha Psi.

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Public Relations in Action.—The Upper Arlington Board of Health passed a resolution of appreciation to Dr. Willard F. Guard, Columbus, at its meeting May 8, 1958. A veteran member of the board, he resigned his post to accept a teaching assignment in India.

Convinced that there should be far more part-time health commissioners or deputy health commissioners who are local veterinarians, Dr. Guard became a member of the board in 1945, at a nominal fee to merely cover expenses. Two years later, he resigned and was reappointed convincing the board members that the work was of such importance that a team of two part-time veterinarians at twice his initial salary was needed. At present, this system is working out well.

Largely due to his efforts, a comprehensive code of health regulations has been adopted by the board and an inspection of food establishments in the city was inaugurated. In 1952, Dr. Guard resigned from the board to become deputy health commissioner in order to put the new plan into action.

Dr. and Mrs. Guard will leave in July for India where he will teach Veterinary Surgery at the College of Rajasthan, a new government sponsored school at Bikaner, India. This project is sponsored jointly by Ohio State University and the United States government. The Guards will go by way of Europe and return by the Pacific in March, 1960.

Pennsylvania

University of Pennsylvania Offers First Postgraduate Course in Bovine Medicine.—The first postgraduate course offered by the School of Veterinary Medicine, University of Pennsylvania, held late last spring, was designed to foster a physiological approach to the practice of bovine medicine and to direct attention to the recent advances made in the field.

The course was strongly oriented toward fundamental understanding of internal medicine. Economically important diseases such as mastitis, leptospirosis, vibriosis, parturient

Participants in the first postgraduate course in bovine medicine held at the School of Veterinary Medicine, University of Pennsylvania, were (standing, left to right)—Dr. A. M. Langdon, Meadville, Pa.; Dr. R. L. Booth, Middleburg, Va.; Dr. W. P. Doherty, Bridgeton, N.J.; Dr. L. S. Nilsson, Jr., Branchville, N.J.; Dr. A. A. Gruen, Chambersburg, Pa.
Seated (left to right)—Dr. M. Feldman, West Chester, Pa.; Dr. S. T. Kashin, Bangor, Maine.

paresis, H-S complex, and mucosal disease were particularly emphasized. It also covered pertinent aspects of neurology, cardiology, parasitology, endocrinology, fluid balance, clinical microscopy, and clinical chemistry.

The school plans to include a second postgraduate course in bovine medicine in its 1959 postgraduate program.

Texas

Drs. Brock and Dickinson Honored.—A testimonial dinner was given by the Dallas Veterinary Medical Association on July 15, 1958, in honor of Dr. W. G. Brock and Dr. R. T. Dickinson of Dallas in recognition of their many years of service in the interests of veterinary medicine and the veterinary profession in their community and state, and on the occasion of their retirement from active practice.

The dinner, held at the Statler Hilton Hotel in Dallas, was attended by a large number of friends of the men honored. Each was presented with a plaque in token of their outstanding service.

Washington

State Association to Meet in Olympia.—The annual meeting of the Washington State V.M.A. which will be held in Olympia, September 8-10, 1958, is expected to attract a wide attendance of veterinarians and their wives from areas throughout the Northwest. Program chairman, Dr. Fred M. Shigley has assembled the program participants from the various fields of practice, from state and national agencies, and from Canada.

The arrangements for the meeting, which will be held in the Olympian Hotel, are sponsored jointly by local, state, and federal veterinarians and their wives. Preceding the regular sessions which begin on Monday morning, September 8, there will be an informal get-together on Sunday afternoon, registration will open, and the association's executive board will meet.

A wide variety of program topics will be presented in the form of papers, forums, and panel discussions. Exhibits will also be featured.

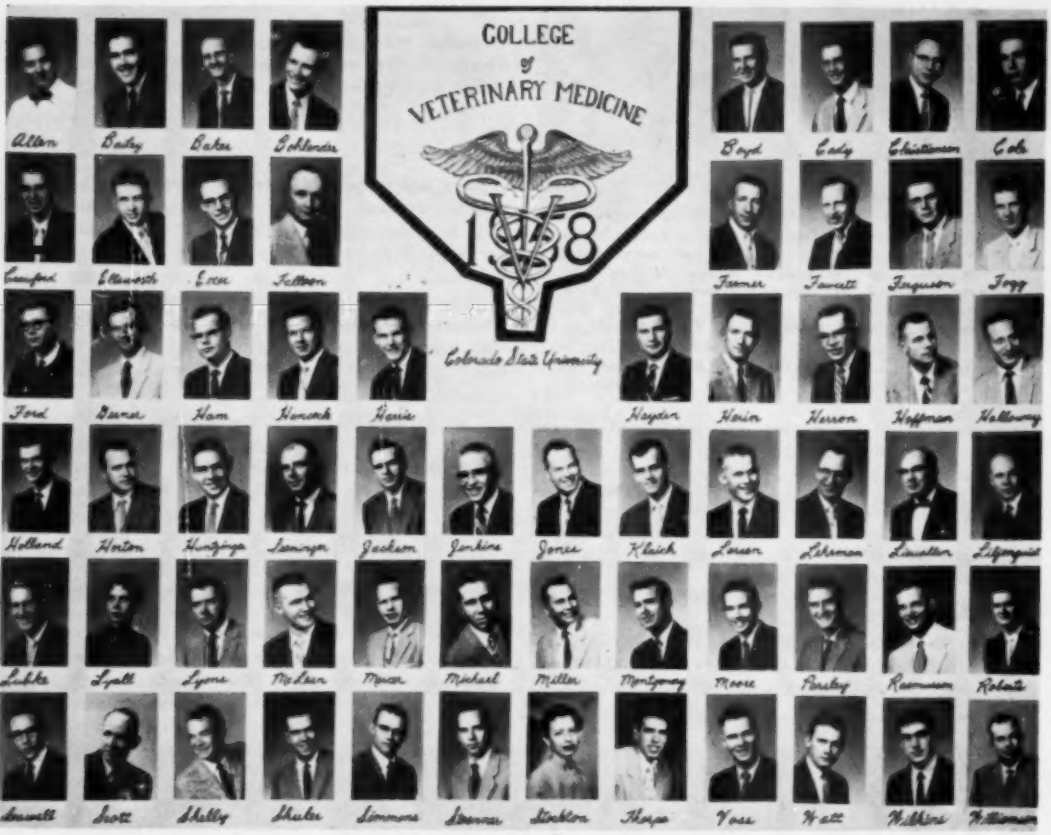
S/IRWIN ERICKSON, *Publicity Chairman.*

The college of Veterinary Medicine choir, State College of Washington, composed entirely of students in the College of Veterinary Medicine, has won the annual song fest at the college four years in succession, in competition with all the campus dormitories, fraternities, and various other groups residing there.

(The commencement pictures and listings of graduates not published in previous issues or in the following pages will appear in the September 1, 1958, issue.)

COMMENCEMENTS

Graduating Class, 1958, College of Veterinary Medicine, Colorado State University



Colorado State University.—At the 1958 commencement exercises of the College of Veterinary Medicine, Colorado State University, the following 59 candidates were presented for the D.V.M. degree:

Robert E. Allen
Robert B. Bailey
John W. Baker
Robert E. Bohlender
Laurence J. Boyd
A. Edward Cady
Dale Christiansen
Walter W. Cole
Richard A. Crawford
Jay P. Ellsworth
Milton W. Etter
Richard A. Falloon
Darrel D. Farmer
Marr O. Fawcett
Benjamin R. Ferguson
Tage J. Fogg
William C. Ford
Charles L. Gerner
Donald J. Ham
William F. Hancock
Charles W. Harris
Ralph J. Hayden
Reginald A. Herin
Kenneth E. Herron
James E. Hoffman
Laurence M. Holland
Delbert V. Holloway
Robert W. Horton
John T. Huntzinger
Howard J. Iseminger
Melvin Samuel Jackson
C. Heyward Jenkins, Jr.
Pierre James Jones
George W. Klaich
Calvert Therald Larson
Charles H. Lehrman
Frank L. Lieuallen
Ira J. Liljenquist
Robert J. Luebke
Mrs. Sonata Manulani Lyall
Douglas McLean
Thomas D. Mercer
Harry A. Michael
Carl W. Miller
Raymond Montgomery
Garland H. Moore
Richard L. Parsley
Jack D. Rasmusson
Richard M. Roberts
Edwin G. Scott
Albert C. Seawell
Noel Barrett Shuler
Robert E. Simmons
Nellie L. Stockton
Robert T. Thorpe
James L. Voss
Thomas L. Watt
Kaye Evan Wilkins
William E. Williamson

Graduating Class, 1958, School of Veterinary Medicine, University of Georgia

Front row (left to right)—H. R. Smith, W. S. Alexander, C. H. Buchan, R. R. White, R. H. Williams, K. L. Huggins, A. E. Dreesen, H. G. Temple, W. E. Salley, H. F. Stolz, R. H. Kemmerlin, J. M. Springs, Jr.

Second row—C. T. Rainey, Jr., J. M. Kelly, B. J. Herlovich, M. H. Boring, J. F. Drawdy, M. W. Livingston, T. H. Eleazer, C. N. Dobbins, Jr., B. F. Miller, T. J. Shirley, Jr., R. H. Batchelor.

Third row—B. H. Wingfield, M. H. Crump, S. A. Ewing, H. T. McDaniel, Jr., D. H. Parker, B. F. Redmon, C. L. Parker, P. M. Morgan, G. C. Edwards, R. C. Elam, H. M. Tate, L. J. Cole, Jr., J. W. Sample, Jr.

Fourth row—P. Freer, M. H. Hatcher, T. C. Gowen, Jr., G. A. McElmurray, Jr., R. Stewart, D. O. Beusse, Jr., W. M. Bryan, M. P. Mackay-Smith, J. E. Awalt, H. Van Lundy, W. Brooks, J. F. Arnold, Jr.

Fifth row—S. L. Arey, R. B. Albritton, T. G. Westmoreland, J. E. Kittrell, J. E. Ducey, E. M. Sink, H. B. Daniel, Jr., C. L. Cannon, D. C. Dunbar, H. E. Walburg, Jr., Columbus Bowen Parsons.

Five graduates not included in the above picture are: C. D. Bird, Jr., W. T. Gerard, C. C. Gurley, H. A. Jordan, A. L. Walker.

University of Georgia.—At the 1958 commencement exercises of the School of Veterinary Medicine, University of Georgia, the following 64 candidates were presented for the D.V.M. degree:

Rayford Burton Albritton
Walker Smith Alexander
Shelvin Lee Arey
James Fred Arnold, Jr.
John Edgar Awalt
Robert Henry Batchelor
Diedrich Oscar Beusse, Jr.
Carl Drayton Bird, Jr.
Matthew Herman Boring
Wayne Brooks
William Melton Bryan
Charles Huntington Buchan
Calvin Lawrence Cannon
Lawrence James Cole, Jr.
Malcolm Hart Crump
Henry Broadus Daniel, Jr.
Charles Nelson Dobbins, Jr.
James Franklin Drawdy
Alice Elizabeth Dreesen
James Emmett Ducey
Dave Clayton Dunbar
George Cleveland Edwards
Raymond Charles Elam
Thomas Henry Eleazer
Sidney Alton Ewing
Paul Freer
William Thomas Gerard
Thomas Cummings Gowen
Carroll Cochran Gurley
Milton Harris Hatcher
Bobby J. Herlovich
Kenneth Lawton Huggins
Harry Alton Jordan
Jacob Martin Kelly
Rudolph Heaner Kemmerlin
John Edison Kittrell
Marvin Whetstone Livingston
Herbert Van Lundy
Matthew Page Mackay-Smith
Hugh Thomas McDaniel, Jr.
Grady Anderson McElmurray, Jr.
Bayne Elmo Miller
Patrick Monroe Morgan
Cleveland Lanier Parker
David Hance Parker
Columbus Bowen Parsons
Cullen Thomas Rainey, Jr.
Billye Faye Redmon
William Everette Salley
John Wendell Sample, Jr.
Thurston Joseph Shirley, Jr.
Early Maxwell Sink
Harvey Roberts Smith
John Morgan Springs, Jr.
Roy Stewart
Hal Fisher Stolz
Hal Manson Tate
H. Glen Temple
Harry Everett Walburg, Jr.
Aubry Lebron Walker
Theodore Garmon Westmoreland
Robert Ruhland White
Ronald Howard Williams
Billy Hillman Wingfield

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University of Illinois.—At the 1958 commencement exercises of the College of Veterinary Medicine, University of Illinois, the following 30 candidates were presented for the D.V.M. degree:

Bernie Allen
Charles Anderson
Le Roy Biehl
Marcus Brown
George Buchal
Norman Cornelius
William Davis
Robert Fetzner
Jay Firth
Robert Goodin
John Harris
Jorg Hoogeweg
Allen Joesten, Jr.
Betty Kjellstrom
Theodore Kjellstrom
Albert Koltveit
Joseph Leveque
Nancy Leveque
Bill Lovett
Lyle McCullough
Jerrold Michaels
Charles Miner
James Pulliam
Frank Riley
Richard Schiltz
John E. Schmidt
Alan Springer
Edward Symonds
Paul Trovillion
Robert C. Willand

(*University of Illinois commencement picture on page 240*)

Graduating Class, 1958, College of Veterinary Medicine, University of Illinois

Top row (left to right)—Jerrold Michaels, Alan Springer, Jay Firth, William Davis, James Pulliam, Albert Koltveit, Jr., Frank Riley, Marcus Brown.

Second row—John Harris, Paul Trovillion.

Third row—Joseph Leveque, Nancy Leveque, Dean Carl A. Brandly, Betty Kjellstrom, Theodore Kjellstrom.

Fourth row—Edward Symonds, Robert Fetzner, Norman Cornelius, Robert Goodin, Charles Miner, Bernie Allen, Jorg Hoogeweg, John E. Schmidt.

Fifth row—Charles Anderson, Lyle McCullough, Robert C. Willand, Allen Joesten, Jr., Richard Schitz, LeRoy Biehl, George Buchal.

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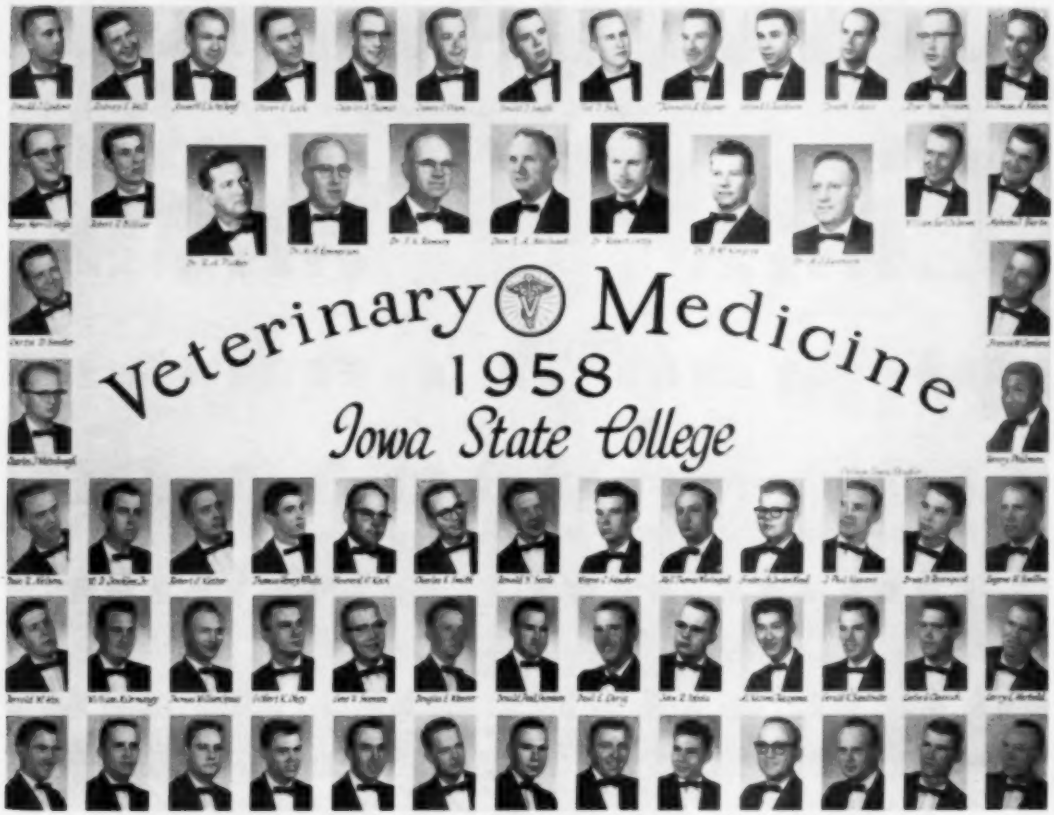
Iowa State College.—At the 1958 commencement exercises of the Division of Veterinary Medicine, Iowa State College, the following 60 candidates were presented for the D.V.M. degree:

James J. Ahern
Melvin J. Barta
Teddy D. Bek
John D. Berthelsen
Robert R. Billiar
Lawrence H. Birchmier
Warren E. Bohnhoff
John D. Clark
Leslie G. Classick
Francis W. Copeland
William M. Cormaney
Joseph Cutaio
William E. DeZeeuw
Paul E. Ehrig
Lowell R. Fahrni
Rodney E. Hall
Larry L. Herbold
Jerrold W. Hix
Roger M. Hogle
Gifford S. Jacobsen
William D. Jenkins, Jr.
Robert G. Ketter
Howard O. Koch
Donald J. Lantow
Craig L. Larson
Herbert A. Lederer
Oliver C. Lock
Dale W. Longtin
Dale R. Nelson
Hillman A. Nelson
Gilbert K. Okey
James O. Olson
Frederick L. Peacock, Jr.
Henry Philmon
Joel L. Prince
Kenneth R. Rayner
Bruce D. Rosenquist
Gerald K. Sandbulte
Curtis D. Sander
Wayne C. Sander
Eugene H. Scallon
Russell L. Schelkopf
Ronald Y. Sevde
Donald P. Shannon
J. Phillip Slavens
Charles E. Smith
Donald D. Smith
Thomas W. Speas
Richard W. Steckelberg
Gene H. Swenson
Al Takayama
Charles A. Thomas
John R. Tobola
Roger Van Prooien
Charles J. Watsabaugh
Douglas E. Weaver
Thomas H. White
Fred J. Wood
Mell T. Wostoupal
Mark F. Young

Graduating Class, 1958, Division of Veterinary Medicine, Iowa State College



Top row (left to right)—Donald J. Lantow, Rodney E. Hall, Russell L. Schelkopf, Oliver C. Lock, Charles A. Thomas, Donald D. Smith, Ted D. Bek, Kenneth R. Rayner, Gifford S. Jacobsen, Joseph Cutaia, Roger Van Prooien, Hillman A. Nelson.

Second row—Roger Merrill Hogle, Robert R. Billiar, Dr. R. A. Packer, Dr. M. A. Emmerson, Dr. F. K. Ramsey, Dean I. A. Merchant, Dr. Robert Getty, Dr. B. W. Kingrey, Dr. M. J. Swenson, William Earl DeZeeum, Melvin J. Barta.

Third row—Curtis D. Sander, Francis W. Copeland.

Fourth row—Charles J. Watsabaugh, Henry Philmon.

Fifth row—Dale R. Nelson, W. D. Jenkins, Jr., Robert G. Ketter, Thomas Henry White, Howard O. Koch, Charles E. Smith, Ronald Y. Sevde, Wayne C. Sander, Mell Thomas Wostoupal, Frederick Jordon Wood, J. Phillip Slavens, Bruce D. Rosenquist, Eugene H. Scallon.

Sixth row—Jerrold W. Hix, William M. Cormaney, Thomas Williams Speas, Gilbert K. Okey, Gene H. Swenson, Douglas E. Weaver, Donald Paul Shannon, Paul E. Ehrig, John R. Tobola, Al Kazumi Takayama, Gerald K. Sandbulte, Leslie G. Classick, Larry L. Herbold.

Seventh row—Dale W. Longtin, Craig L. Larson, Herbert A. Lederer, Joel L. Prince, Lawrence H. Birchmier, Frederick L. Peacock, Jr., John D. Berthelsen, James John Ahern, Mark F. Young, Lowell Robert Fahrni, Warren E. Bohnhoff, R. Wayne Steckelberg, John D. Clark.

Graduating Class, 1958, College of Veterinary Medicine, Michigan State University

Top row (left to right)—Ellin C. Abbott, Lawrence Ackerman, Dean W. Anderson, Richard L. Asquith, William H. Ball, William A. Barber, Eldon C. Barclay, Jr., James N. Boydston, Gerald Brown, Morley N. Burns, John D. Cardno, Ann N. Clark.

Second row—Alpha S. Clark, Frank J. Coussens, Joseph F. Curley, Earle M. Davis, Jr.

Third row—Chester G. DeJongh, Elbridge L. Dunckel, David V. Engstrom, A. C. Fields, Jr., Phillip M. Flora, Robert F. Goodbary, Richard S. Grace, Russell A. Greene.

Fourth row—John E. Greve, George D. Harris, James M. Harris, James L. Harrison, Walter R. Harthun, Dean W. W. Armistead, Thomas N. Heslip, Edward J. Hinsman, Robert L. Humphreys, Arthur A. Jarvis, James E. Keel.

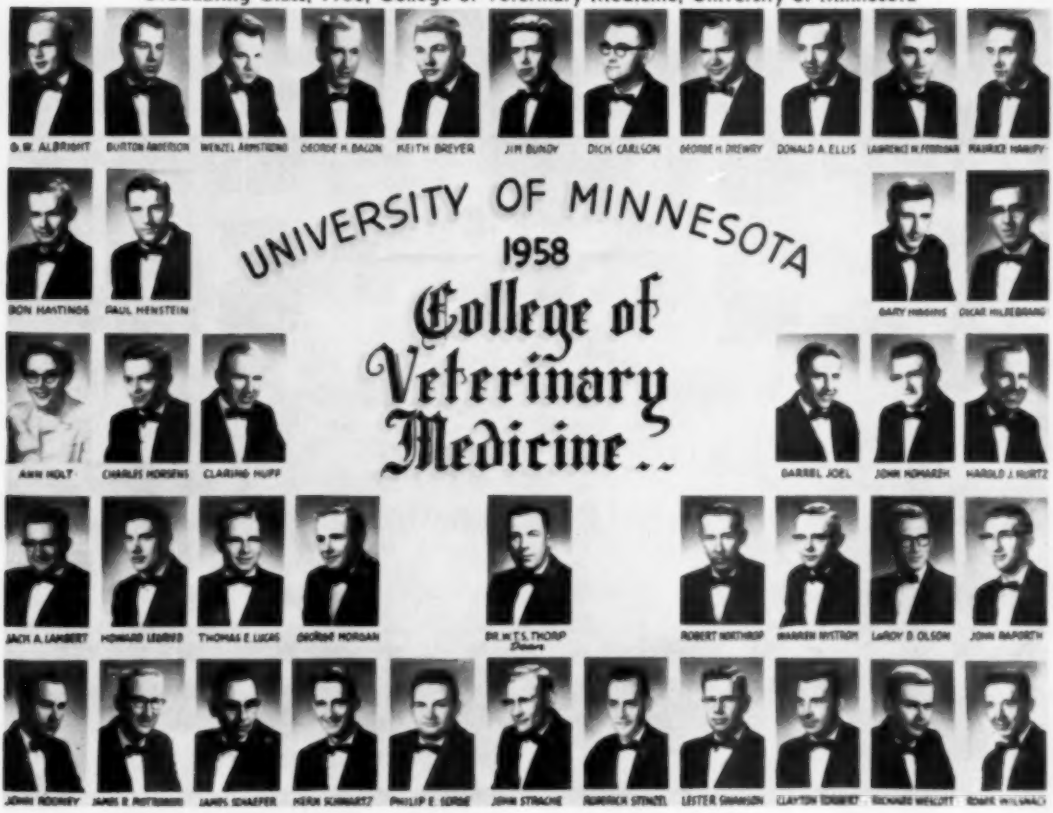
Fifth row—Rolla J. Keeran, Gerald J. Kugel, Aaron M. Leash, George J. Lombard, Charles R. Martin, Jack A. Martin, William E. Morrill, Ulreh V. Mostosky, Richard F. Nokes, J. Wayne O'Connell, Kenneth D. Russell, Edward Sadilek.

Sixth row—Wayne E. Salsbery, Fred B. Shulak, Norris H. Signs, Mary E. Smith, James H. Staples, Donald G. Turner, Sally A. Turner, J. Vanniasingham, Ian A. Wallace, James White, Wayne A. Wickert, Sr., Lloyd H. C. Wong.

Michigan State University.—At the 1958 commencement exercises of the College of Veterinary Medicine, Michigan State University, the following 58 candidates were presented for the D.V.M. degree:

Ellin C. Abbott
Lawrence A. Ackerman
Dean W. Anderson
Richard L. Asquith
William H. Ball
William A. Barber
Eldon C. Barclay, Jr.
James N. Boydston
Gerald Brown
Morley N. Burns
John D. Cardno
Alpha S. Clark
Ann N. Clark
Frank J. Coussens
Joseph F. Curley
Earle M. Davis, Jr.
Chester G. DeJongh
Elbridge L. Dunckel
David V. Engstrom
Alexander Fields, Jr.
Philip M. Flora
Robert F. Goodbary
Richard S. Grace
Russell A. Greene
John H. Greve
George D. Harris
James M. Harris
James L. Harrison
Walter R. Harthun
Thomas N. Heslip
Edward J. Hinsman
Robert L. Humphreys
Arthur A. Jarvis
James E. Keel
Rolla J. Keeran
Gerald J. Kugel
Aaron M. Leash
George J. Lombard
Charles R. Martin
Jack A. Martin
William E. Morrill
Ulreh V. Mostosky
Richard F. Nokes
John W. O'Connell
Kenneth D. Russell
Edward Sadilek
Wayne E. Salsbery
Fred B. Shulak
Norris H. Signs
Mary E. Smith
James H. Staples
Donald G. Turner
Sally A. Turner
John A. Vanniasingham
Ian A. Wallace
James White
Wayne A. Wickert, Sr.
Lloyd H. C. Wong

Graduating Class, 1958, College of Veterinary Medicine, University of Minnesota



University of Minnesota.—At the 1958 commencement exercises of the College of Veterinary Medicine, University of Minnesota, the following 40 candidates were presented for the D.V.M. degree:

G. W. Albright
Burton Anderson
Wenzel Armstrong
George K. Bacon
Keith Breyer
Jim Bundy
Dick Carlson
George H. Drewry
Donald A. Ellis
Lawrence W. Ferrigan
Maurice Hanify
Don Hastings
Paul Henstein
Gary Higgins
Oscar Hildebrandt
Ann Holt
Charles Horsens
Claring Huff
Darrell Joel
John Komarek
Harold J. Kurtz
Jack A. Lambert
Howard Legried
Thomas E. Lucas
George Morgan
Robert Northrop
Warren Nystrom
LeRoy D. Olson
James R. Piotrowski
John Raforth
John Rooney
James Schaefer
Kern Schwartz
Philip E. Sorge
John Strache
Roderick Stenzel
Lester Swanson
Clayton Torbert
Richard Wescott
Roger Wilsnack

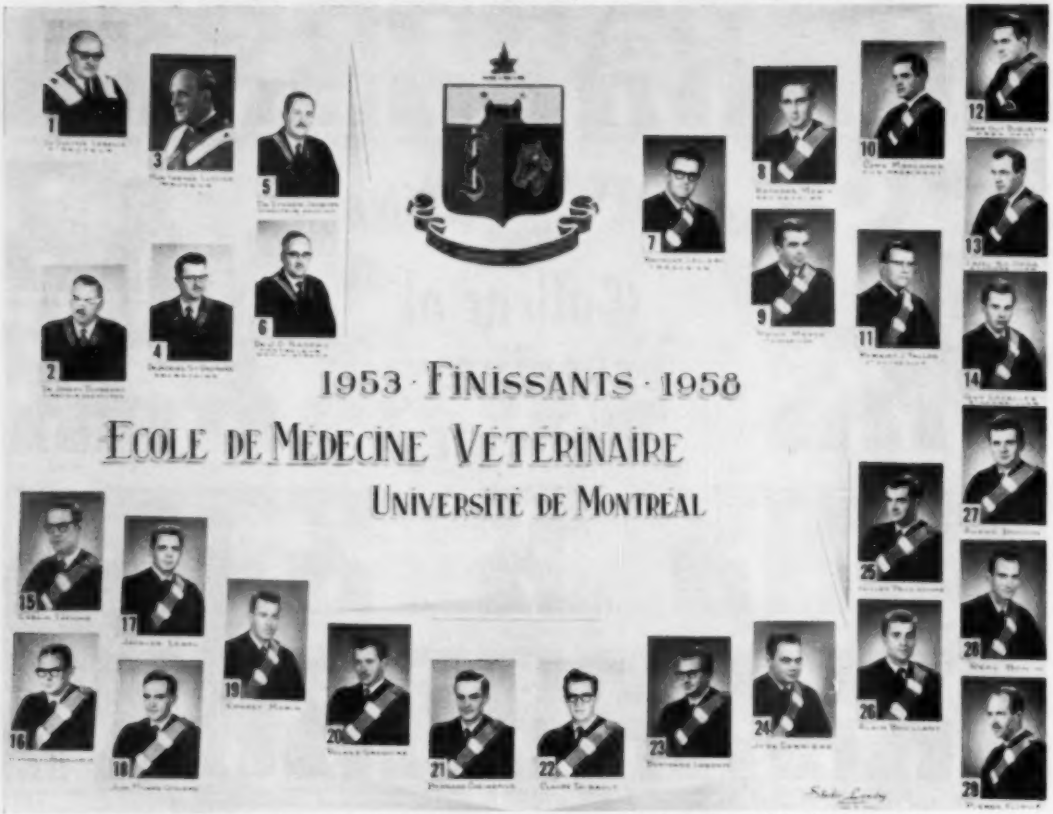
• • •

University of Montreal.—At the 1958 commencement exercises of the School of Veterinary Medicine, University of Montreal, the following 23 candidates were presented for the D.V.M. degree:

Fernando Abbruzzese
Andre Bohuon
Real Bonin
Alain Bouillant
Jude Carriere
Bernard Chevrefils
Jean-Guy Duquette
Robert J. Fallon
Pierre Flipot
Jean-Pierre Giguere
Roland Gregoire
Bertrand Labonte
Guy Lavallee
Urbain Lavigne
Jacques Lebel
Raymond Leclerc
Come Marchand
Rene Masse
Ernest Morin
Raymond Morin
Gilles Prud'Homme
Laval Saint-Onge
Claude Thibault

(*University of Montreal commencement picture on page 244*)

Graduating Class, 1958, School of Veterinary Medicine, University of Montreal



Those in the picture are (1) Dr. Gustave Labelle, (2) Dr. Joseph Dufresne, (3) Mgr. Irenée Lussier, (4) Jacques St. Georges, (5) Dr. Ephrem Jacques, (6) Dr. J. D. Nadeau, (7) Raymond Leclerc, (8) Raymond Morin, (9) Rene Masse, (10) Come Marchand, (11) Robert J. Fallon, (12) Jean-Guy Duquette, (13) Laval Saint-Onge, (14) Guy Lavallee, (15) Urbain Lavigne, (16) Fernando Abbruzzese, (17) Jacques Lebel, (18) Jean-Pierre Giguere, (19) Ernest Morin, (20) Roland Gregoire, (21) Bernard Chevrefils, (22) Claude Thibault, (23) Bertrand Labonte, (24) Jude Carriere, (25) Alain Bouillant, (26) Andre Bohuon, (27) Real Bonin, (28) Pierre Flipot.

• • •

Oklahoma State University.—At the 1958 commencement exercises of the College of Veterinary Medicine, Oklahoma State University, the following 34 candidates were presented for the D.V.M. degree:

Abdul Amir Jabir Al-Hummadi
Jerry M. Bonham
Tommy James Byrd
Samuel Forrest Cheesman
John H. Collins
Clement V. Cottom, Jr.
Donald R. Crockett
John Williamson Davis, Jr.
Murtadha Hashim El-Hashimi
Lawrence Hardy Erwin
Rex Richard Every
Robert Lee Godby
Louis Wells Heavner, Jr.
Buddie Curtis Hickey
Joe Ben Jolliffe
Paul L. Kunneman
Thomas Robert Latham
Robert Lee Lawrence
George Arnold Lester
William R. McCallon
Elmer Edward McCrary
Kermit Wayne Minton
Charles Earnest Wyatt
Thornal Durant O'Quinn, Jr.
John Cecil Peckham
Jack Wayne Peterson
Harold B. Rinker
Alfred Allison Robinson
John McNeil Rust
David Lloyd Seaman
James Gordon Sewell
Robert McFeters Spragg
Wayne Eugene Stout
Lloyd Charles Weldon

Graduating Class, 1958, College of Veterinary Medicine, Oklahoma State University

Top row (left to right)—L. C. Weldon, D. L. Seaman, J. G. Sewell, C. E. Wyatt, J. M. Rust, A. A. Robinson, J. C. Peckham.

Second row—R. M. Spragg, W. E. Stout, H. B. Rinker, K. W. Minton, E. E. McCrary, W. R. McCallon, T. D. O'Quinn, Jr.

Third row—J. W. Peterson, G. A. Lester.

Fourth row—R. L. Lawrence, T. R. Lathan, P. L. Kunneman, L. H. Erwin, R. L. Godby, J. B. Jolliffe.

Fifth row—B. C. Hickey, D. R. Crockett, M. H. El-Hashimi, J. W. Davis, Jr., R. R. Every, L. W. Heavner, Jr.

Sixth row—T. J. Byrd, A. A. Al-Hummadi, J. M. Bonham, S. F. Cheesman, J. H. Collins, C. V. Cottom, Jr.

Graduating Class, 1958, School of Veterinary Medicine, A. & M. College of Texas

Two graduating seniors who do not appear in the above picture are: Dr. Frank M. Roach and Dr. Albert L. Burroughs.

A. & M. College of Texas.—At the 1958 commencement exercises of the School of Veterinary Medicine, A. & M. College of Texas, the following 62 candidates were presented for the D.V.M. degree:

Sid Allen
Donald J. Anderson
Richard L. Baker
B. W. Blakewood
A. L. Burroughs
D. R. Cardwell
Lamar H. Carroll
Roy G. Chaney
James E. Clark
Dan J. Craig
Delbert E. Dawson, Jr.
Don H. Dixon
Thomas W. Dorrell
G. A. Garza
A. J. Grezaffi
Fred Hartman
James B. Henson
Wm. A. Hill
Dan W. Hilliard
T. F. Hubert
John W. Huff
R. B. James, II
Grady R. Kane
J. T. LaCroix
John K. Lassig
R. A. Lee
J. D. Loftis
Burch Loggins
R. S. McCluer
James D. McCrady
J. H. McCrary
Derry D. Magee
Frank E. Mann, Jr.
J. H. Mead, III
J. G. Netardus
V. H. Patrick
Bill Perry
W. B. Porter
Johnny Putty
J. D. Quinn
R. C. Rabe
J. T. Reyman
S. L. Reynolds
Jerry Rheudasil
F. M. Roach
Tom W. Rose
Gus W. Salbador
C. T. Schenck
O. C. Schlinke
Wayne T. Slone
Jimmy B. Smith
John Paul Smith, Jr.
J. S. Stanton
V. Q. Telford
D. R. Turbeville
J. R. Vriesenga
Wm. C. Wafer
J. W. Watters
T. D. Williams
Charles F. Wilson
Jim Witcher
R. T. Wyatt

DEATHS

Star Indicates Member of AVMA

★**Robert L. Anderes.**—Dr. Robert Louis Anderes, widely known editor and publisher of *Veterinary Medicine*, died from a heart attack on July 21, 1958, while attending a meeting in Chicago.

Dr. Robert L. Anderes

Born July 14, 1902, at Kansas City, Kan., Dr. Anderes attended the local public schools and Kansas State College, receiving the B.S. degree in 1925 and the D.V.M. degree in 1934. During the following two years, he was associated with Dr. J. V. Lacroix in the practice of veterinary medicine at the North Shore Animal Hospital in Evanston, Ill. From 1936 to 1940, Dr. Anderes was on the professional staff of Jensen-Salsbery Laboratories at Kansas City and then was on active duty with the Army Veterinary Corps from 1940 to 1946 in which he attained the rank of colonel and remained on the reserve status following separation from active duty.

In 1946, he again entered practice and operated a small animal hospital in Kansas City until 1949. In that year, Dr. Anderes acquired *Veterinary Medicine* and The Veterinary Medicine Publishing Company from the estate of Dr. D. M. Campbell, long-time editor and publisher of *Veterinary Medicine*. During the nine years of Dr. Anderes' editorship, *Veterinary Medicine* maintained its high standards and improved its format. It is a distinct credit to the veterinary medical profession.

He was a member of the Missouri, Kansas, and Kansas City (past-president) veterinary medical associations, the American Animal Hospital Association, the Society for Animal Production, U.S. Livestock Sanitary Association, U.S. Committee of the XVth and the XVIth International Veterinary Congresses, and had been a member of the AVMA for 22 years. He was also a member of Sertoma International and of St. Andrew's Episcopal Church in Kansas City. He had served many years as a scoutmaster.

Among the honors to his credit was membership in Phi Kappa Phi. In 1957, he received the Distinguished Service Award presented annually by his alma mater, Kansas State College.

He is survived by his wife, Mrs. Catherine Busey Anderes; a son, Robert L. Anderes, Jr.; a daughter, Kay Sandra Anderes; and his mother, Mrs. Otto Anderes.

Dr. Anderes was a popular speaker, wrote many technical papers, and favorably influenced both the standards and the prestige of veterinary literature and journalism. It can be said that he served his country and his profession well.

• • •

John G. Blum (CVC '08), 74, Pomona, Calif., formerly a resident of Normal, Ill., died suddenly May 27, 1958, at his daughter's home in Ellsworth, Ill., while visiting there.

Dr. Blum had practiced in Normal from 1908 to 1945. He then retired and moved to California. Among other affiliations, Dr. Blum was a member of the Normal Masonic Lodge, Normal Eastern Star Chapter, the Al Malaikah Shrine of Los Angeles, Calif., and the Pomona Optimist Club. He was also a past-president of the Illinois V.M.A.

Surviving are his widow, Elizabeth Miller Blum, three daughters, two sisters, and three half brothers.

William Bowerman, 67, Rogers, Ark., died May 3, 1958, at the Rogers Memorial Hospital following an illness of nine months.

Dr. Bowerman had practiced in Rogers and Springdale, Ark., for about 30 years before his retirement. He is survived by his widow, Grace Bowerman, two daughters, and a son.

★**Paul A. Carlson** (MSC '43), 46, Fresno, Calif., died May 21, 1958, from lymphosarcoma.

Dr. Carlson was the district veterinarian in five central California counties for the bureau of livestock disease control, State Department of Agriculture. He supervised the work of ten veterinarians in the Fresno office, and had been with the state bureau in Fresno for six years. Previously, he had operated his own hospital in San Diego.

A member of the California V.M.A., he was also a past-president of the Central California V.M.A. He is survived by his widow, Carolyn, three children, his mother, and a brother.

Carey Roy Conger, 82, Escanaba, Mich., director of the Seventh-Day Adventist Center in Escanaba, died May 20, 1958, at his home.

Retired from practice, Dr. Conger was also a teacher and farmer. He was born in Saybroch, Ill., and lived in Skandia, Mich., before moving to Escanaba five and a half years ago.

Surviving Dr. Conger are his widow, Alpha Carlson Conger, a brother, and two sisters.

John Stanley Craft (KCV '14), 72, Ottumwa, Iowa, died at the Ottumwa Hospital on May 27, 1958, after suffering a stroke two days previously.

Surviving Dr. Craft are his widow, Flora, three brothers, four sisters, and several nieces and nephews.

★**Walter S. Craig** (KCV '10), 76 Montrose, Colo., died recently according to a notice received by the Association in June, 1958, from the post office.

Dr. Craig was made a life member of the AVMA in 1956.

Fred Erdel (CVC '05), 76, Mulberry, Ind., died May 16, 1958, following an illness of 16 years.

Dr. Erdel had practiced in Clinton County from the time of his graduation up until the time of his retirement in 1942. He is survived by his widow, Hazel Harrell Erdel.

James J. Hamiter, 81, Dallas, Texas, the oldest practicing veterinarian in the state, died May 9, 1958, in his home, following a heart attack.

In a career that spanned more than 50 years, Dr. Hamiter specialized in lions, tigers, leopards, and cheetahs. In addition to his private practice, he served as the Dallas zoo's veterinarian for 30 years. He treated the animals for almost half that time without charge. Dr. Hamiter retired as zoo veterinarian two years ago but continued in his own practice.

He is survived by two sons, a brother, and two great-grandchildren.

Rolph M. Jenkins (ONT '07), Don Mills, Ont., former Canadian national exhibition director and manager of Don-Alda Farms, died May 11, 1958, at his home in Don Mills.

Dr. Jenkins managed the Don-Alda Farms for 30 years. The farms, a 600-acre agricultural showplace, were visited by farm experts from all over the world.

He is survived by his widow, Isabel Wray Jenkins, a daughter, and a son.

★**Arthur L. Leibert** (UP '38), 46, Center Valley, Pa., died suddenly in his home on May 30, 1958, after becoming ill during an airplane flight over the Lehigh Valley. He was taken to his home where he died a short time later.

Dr. Leibert completed his preveterinary studies at Moravian College in 1934 and received his D.V.M. degree from the University of Pennsylvania in 1938. He did postgraduate work in Utrecht, Netherlands.

A member of the Pennsylvania V.M.A. and of the Lehigh Valley V.M.A., Dr. Leibert was also affiliated with Greenleaf Masonic Lodge, Allentown, and the Lehigh Consistory. He is survived by his widow, Catherine E. Wuchter Leibert, and a son.

Lawrence Lewis (CVC '09), 74, Celina, Texas, died May 21, 1958, in his home after several years of illness.

Surviving Dr. Lewis are his widow, Gertrude Brent Lewis, a daughter, a brother, and a sister.

P. C. Lindeman, 81, Red Lion, Pa., died May 17, 1958, at his home.

Born in Drums, Pa., Dr. Lindeman was employed by the Pennsylvania State Bureau of Animal Husbandry prior to his retirement.

He is survived by his widow, the former Anna Payne, a brother, and a sister.

R. L. Peterson (KCV '12), 70, Polk, Neb., died in Bryan Memorial Hospital where he had undergone surgery four days previously. He had been in failing health for eight years.

Dr. Peterson had practiced in Polk from the time of his graduation up until his health began to fail. His hobbies were his garden and rare birds of which he had more than 200 including pheasants and peacocks. He served as director of the Polk band for 26 years.

He is survived by his wife, Lydia Erickson Peterson, a son, a brother, and a sister.

Robert L. Rogers, Jr., (TEX '25), 55, Fort Worth, Texas, organizer and president of the Texas Phenothiazine Company, died May 4, 1958, in a local hospital after a short illness.

Dr. Rogers was director of the Texas Livestock Sanitary Commission Laboratory in Fort Worth from 1939 to 1941, and was a former member of the State Board of Veterinary Medical Examiners.

He is survived by his wife, his mother, a sister, and a brother.

Ora H. Stanford (ONT '16), 75, Cambridge Springs, Pa., died May 18, 1958, in Randolph Township, where he had been called to care for a sick animal.

Last June, Dr. Stanford and his wife celebrated their fiftieth wedding anniversary. He was a member of the Richmond Grange and the Teepleville Methodist Church.

Besides his widow, Ruby Lha Bailey Stanford, he is survived by two sons and five grandchildren.

PUBLISHED REPORTS OF EXPERIENCE WITH

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Reprint from the JOURNAL of the American Veterinary Medical Association, vol. 124, No. 922, Jan., 1954, pp. 19-20.

A Note on the Use of Surital Sodium Anesthesia in Swine

H. W. DUNNE, D.V.M., Ph.D., and Captain S. C. BENBROOK, V.C., U.S. Army

East Lansing, Michigan

THE NEED for a satisfactory anesthetic in swine practice has long been recognized by veterinary surgeons and research workers throughout the nation. Until recently, most of the anesthetics available seemed to be contraindicated in some phase of application. They proved to be either toxic, resulting in frequent deaths, or the duration of anesthesia was too long and incurred time-consuming and struggling aspiration. Floundering and struggling associated with recovery from anesthesia of

---

Use of Surital Sodium and Curare in Small-Animal Surgery

W. O. Brinker, D.V.M., M.S.
Michigan State College
East Lansing, Mich.

Reprint, The North American Veterinarian, Vol. 32, December, 1951.

Reprinted from VETERINARY MEDICINE, Vol. XLVII, No. 3, March 1952

Surital Sodium And Morphine Anesthe
In Canine Surgery

W. C. YOUNG, JR.,* D.V.M. and F. E. EADS
D.V.M., M.S., Detroit, Michi

THE use of surital** sodium (sodium 5-allyl-5-(1-methylbutyl)-2-thiobarbiturate), a recently developed ultra-short acting thiobarbiturate, has been reported by several workers. It could be called the thiopentone of sesamal** just as pentothal** sodium could be considered the thiopentone of nembutal**.

In most instances, intravenously. Hunter Dillon and Dymond** administration as re in this manner has very moderate

In five experim

Surital Sodium, A New Anesthetic and Hypnotic

Studies in Dogs

T. F. REUTNER, D.V.M., M.S., and O. M. GRUMZIT, M.S., M.D.

Detroit, Michigan

Reprint from the Journal of the American Veterinary Medical Association, Vol. CXIII, No. 880, October, 1948, pp. 257-260.

Surital Sodium Anesthesia in Canine Surgery

H. B. ROBERTS, D.V.M.; W. E. WENDT, D.V.M.; C. C. WAGNER, D.V.M., M.Sc.; T. F. REUTNER, D.V.M., M.S.

Reprint from the JOURNAL of the American Veterinary Medical Association, Vol. CXVIII, No. 880, March, 1951, pp. 191-192.

Reprinted from VETERINARY MEDICINE, Vol. L, No. 8, p. 365, August 1

Intrathoracic Anesthesia In Cats

MARK STERNFELS, D.V.M., Mount Vernon, New York

WHEN a human patient is suffering from an ailment which calls for surgical interference, laboratory technics are employed to determine and solve solutions are given to raise blood sugar levels for prevention of shock and dehydration.

The anesthetist is usually part of the surgical

Surgery performed under anesth by intrathoracic administration clude: Teeth cleaning and ar trimming and removing mal or abscesses, removal of injure tumors, removal of cysts, pe pyometra, castration, cesarea treatments, accident cases, p fractures, and for x-ray diagno

---

Dosage Studies of Surital Sodium in Dogs

ROBERT F. BORGMAN D.V.M., M.S.

Asheville, North Carolina

RECENTLY, a new thiobarbiturate, surital sodium, has been offered for limited clinical trial. Clinical reports have shown surital sodium to be an excellent anesthetic for operations of short duration in dogs. Its anesthesia is smooth and relatively free from excitement and irritability. When injected intravenously at hourly intervals,

RESULTS

A total of 11 dogs were necessary dose necessary for 2.5 mg. of surital sodium pe weight. Of the 2 dogs an

TABLE 1.—Results of Surital So

Surital Sodium Anesthesia in Dogs and Cats
A Symposium Covering 7522 Patients

F. E. EADS, D.V.M., M.S., Detroit, Mich.

Although intravenous anesthesia is one of the oldest means of rendering patients insensible to pain, the method has been actively studied only within the past 15 years. So far as is known, the earliest attempt to produce anesthesia by vein was made in 1665 when a solution of opium was given by means of a

very. They estimate that the approximately 1.5 times that of the dog. Similar results were Kelly, et al.** Surital seems obesity in dogs than does pen there appears to be less saliva toxicholism manifestations whe

(*Reprinted from* THE CALIFORNIA VETERINARIAN, Sept.-Oct., 1953.)

These and other publications on Surital available on request

Department of Veterinary Medicine

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MEDICAMENTA VERA

Dr. Scheidy Re-Elected in District II, Dr. Woelffer New Member from District III

DISTRICT II (DELAWARE, DISTRICT OF COLUMBIA, MARYLAND, NEW JERSEY, PENNSYLVANIA)

In balloting completed on July 12, 1958, Dr. S. F. Scheidy, Bryn Mawr, Pa., was elected to another term in Executive District II. He was first elected to the Board in 1943 and has served continuously since then. He was chairman of the Board in 1954-1955.

A graduate of the University of Pennsylvania, class of 1929, Dr. Scheidy interned at the Vet-

Dr. S. F. Scheidy

erinary Hospital of his alma mater from 1929 to 1931. He was associated with the university veterinary clinical faculty from 1938-1943, either on a full or part-time basis. Currently, he is assistant professor of clinical veterinary medicine and is also veterinary medical director of Smith, Kline & French Laboratories, Philadelphia. He held a similar position with Merck, Sharp and Dohme from 1943 to 1957. At that time, Smith, Kline & French, medical pharmaceutical manufacturers, decided to investigate the application of its products in the field of veterinary medicine and Dr. Scheidy was placed in charge of this work.

DISTRICT III (ILLINOIS, INDIANA, WISCONSIN)

In balloting also completed on July 12, 1958, Dr. E. A. Woelffer, practitioner of Oconomowoc, Wis., was elected to a five-year term in District III. He succeeds Dr. L. M. Hutchings who has been a member of the Board since 1953 and is its present chairman.

Dr. Woelffer is a native of Wisconsin, having been born in Waterloo in 1897. He received the B.S. degree in agriculture at the University of Wisconsin in 1922 after which he showed pure bred dairy cattle for a few years. He entered New York State Veterinary College at Cornell Univer-

Dr. E. A. Woelffer

sity in 1927 and received his D.V.M. degree in 1931. Following graduation, he went with H. P. Hood & Sons Co., certified milk producers of Boston, Mass., as chief veterinarian, a position he held until 1947 when he accepted appointment as extension veterinarian at the University of Illinois.

In 1948, Dr. Woelffer went with Pabst Farms, Oconomowoc, as veterinarian for their dairy herds, then entered private practice in 1950 and has specialized in bovine reproductive diseases. He is widely known among veterinarians and dairy cattle groups, having appeared on many association and conference programs.

He joined the AVMA following graduation, has served on various committees, and is presently a member of the Special Committee on Animal Reproduction and Artificial Insemination.

Drs. N. R. Boothe, E. R. Maschgan, and Jo Anne Schmidt of Chicago served as a board of tellers on July 15 and certified the election of Drs. Scheidy and Woelffer.

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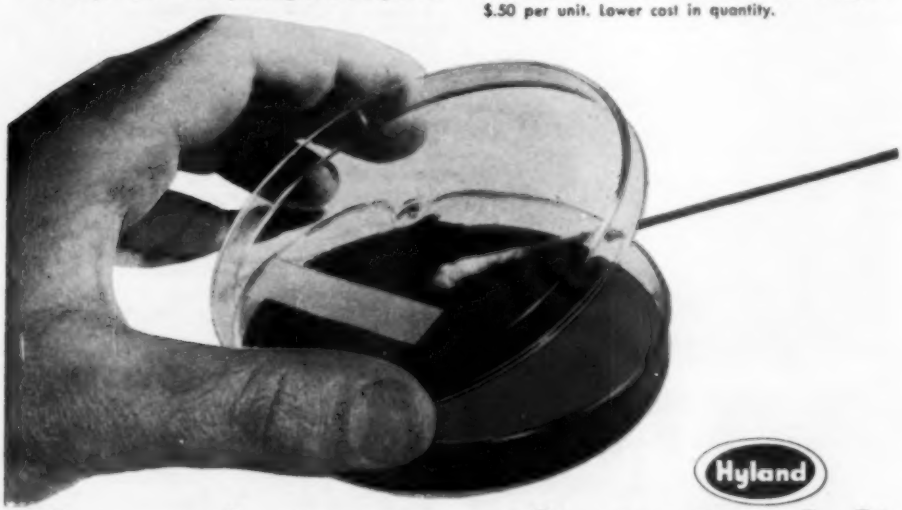

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APPLICATIONS

Graduate Applicants

The following are graduates who have recently received their veterinary degree and who have applied for AVMA membership under the provision granted in the Administrative Bylaws to members in good standing of student chapters.

First Listing

A. & M. College of Texas

CARROLL, LAMAR H., D.V.M.
701 Nall Lane, Bryan, Texas.
Vouchers: J. P. Davis and G. S. Trevino.
CLARK, JAMES E., D.V.M.
Wheeler, Texas.
Vouchers: F. C. Neal and A. A. Lenert.
DORRELL, THOMAS W.
411 Foch St., Bryan Texas.
Vouchers: G. S. Trevino and J. P. Davis.
HUFF, JOHN W., D.V.M.
Box 66, Wenton, Texas.
Vouchers: R. J. Beamer and G. S. Trevino.
LEE, ROBERT A., D.V.M.
2118 Glenn Haven, Houston 25, Texas.
Vouchers: R. T. DuBose and S. M. Gaafar.
McCLUER, ROBERT S., D.V.M.
Box 485, Jena, La.
Vouchers: J. P. Davis and W. M. Romane.
REYNOLDS, SCOTT L., D.V.M.
Box 968, Andrews, Texas.
Vouchers: C. W. Schulz and F. C. Neal.
SLONE, WAYNE T., D.V.M.
Box 1149, Bay City, Texas.
Vouchers: W. M. Romane and P. L. Hubert.
WAFER, WILLIAM C., Jr., D.V.M.
7344 Franklin Rd., El Paso, Texas.
Vouchers: G. S. Trevino and P. L. Hubert.

University of Georgia

ALBRITTON, RAYFORD B., D.V.M.
Rt. 1, Rupert, Ga.
Vouchers: J. D. Edens and J. D. Morton.
BELISSE, DIEDRICH O., Jr., D.V.M.
887 Barnett Pl., N. E., Atlanta, Ga.
Vouchers: T. J. Jones and E. W. Causey.
BORING, MATTHEW H., D.V.M.
Myersville, Md.
Vouchers: E. W. Causey and J. D. Edens.
BROOKS, WAYNE, D.V.M.
RFD 1, Box 40, Sparta, N. Car.
Vouchers: M. Rohleder and T. J. Jones.
BRYAN, WILLIAM M., D.V.M.
Ahoskie, N. Car.
Vouchers: J. D. Morton and E. W. Causey.
BUCHAN, CHARLES H., D.V.M.
Colorado State Univ., Dept. of Veterinary Medicine, Fort Collins, Colo.
Vouchers: E. W. Causey and M. Rohleder.
COLE, LAWRENCE J., Jr., D.V.M.
Gainesville, Fla.
Vouchers: T. J. Jones and J. D. Morton.
DANIEL, HENRY B., Jr., D.V.M.
Rt. 2, South Boston, Va.
Vouchers: T. J. Jones and J. D. Morton.
DRAWDY, JAMES F., D.V.M.
Rt. 1, Lenox, Ga.
Vouchers: T. J. Jones and A. M. Mills.
DREESEN, A. ELIZABETH, D.V.M.
Box 457, Campus Station, Athens, Ga.
Vouchers: T. F. Zweigart and C. Westerfield.
DUCEY, JAMES E., D.V.M.
2321 Bona Venture Dr., Savannah, Ga.
Vouchers: D. Sikes and E. W. Causey.
EDWARDS, GEORGE C., D.V.M.
Rt. 2, Middlesex, N. Car.
Vouchers: J. D. Edens and A. M. Mills.
ELAM, RAYMOND C., D.V.M.
Merrifield, Va.
Vouchers: F. A. Hayes and C. Westerfield.
FREER, PAUL, D.V.M.
Box, Campus Station, Athens, Ga.
Vouchers: J. D. Edens and A. M. Mills.
GERARD, WILLIAM T., D.V.M.
P.O. Box 363, Athens, Ga.
Vouchers: E. W. Causey and T. J. Jones.
GOWEN, THOMAS C., Jr., D.V.M.
Folkston, Ga.
Vouchers: E. W. Causey and L. K. Taul.
HERLOVICH, BOBBY J., D.V.M.
1050 N. W., 116th St., Miami, Fla.
Vouchers: T. J. Jones and A. M. Mills.
HUGGINS, KENNETH L., D.V.M.
Marion Animal Hospital, Marion, S. Car.
Vouchers: T. J. Jones and J. D. Morton.
KEMMERLIN, RUDOLPH H., D.V.M.
Bamberg, S. Car.
Vouchers: D. Sikes and T. J. Jones.
LIVINGSTON, MARVIN W., D.V.M.
4709 Gallatin Rd., Nashville, Tenn.
Vouchers: J. D. Morton and T. J. Jones.
LUNDY, HERBERT V., D.V.M.
Rt. 3, Milton, Fla.
Vouchers: E. W. Causey and T. J. Jones.
MACKAY-SMITH, MATTHEW P., D.V.M.
39th St. & Woodland Ave., Philadelphia 4, Pa.
Vouchers: L. K. Taul and E. W. Causey.
MILLER, BAYNE E., D.V.M.
Rt. 2, Mocksville, N. Car.
Vouchers: J. D. Morton and T. J. Jones.
MORGAN, PATRICK M., D.V.M.
1140 Oak Ave., Panama City, Fla.
Vouchers: J. D. Edens and E. W. Causey.
PARSONS, C. BOWEN, D.V.M.
101 Catherine St., Pickens, S. Car.
Vouchers: J. D. Morton and T. J. Jones.
RAINEY, CULLEN T., Jr., D.V.M.
Ellaville, Ga.
Vouchers: J. D. Edens and A. M. Mills.
SALLEY, W. EVERETTE, D.V.M.
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TURNER, SALLY A., D.V.M.
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164 New Hampshire Ave., London, Ohio.
BARTELS, DONALD R., Sr., D.V.M.
27050 Solon Rd., Solon, Ohio.
BEALL, CHARLES W., D.V.M.
Clarksburg, Ind.
BERKKAMP, ROLLAND D., D.V.M.
Melmore Rd., Tiffin, Ohio.
BOWERS, JOHN F., D.V.M.
3315 Shelby St., Indianapolis, Ind.
BUSEY, WILLIAM M., D.V.M.
141 E. South St., Worthington, Ohio.
BRICKMAN, HOMER W., D.V.M.
325 LaGrange Rd., Elyria, Ohio.
BROWN, LOWELL N., D.V.M.
Department of Veterinary Hygiene, Iowa State College, Ames, Iowa.
BOYER, FREDERICK, E., D.V.M.
Jackson Center, Ohio.
BRIGHT, WILLIAM B., D.V.M.
Converse, Ind.
BURKHOLDER, RALPH H., D.V.M.
Rt. 1, Archbold, Ohio.
BURWELL, RICHARD E., D.V.M.
Danville, Ohio.
BUTHKER, HAROLD L., D.V.M.
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CAIN, JOSEPH I., D.V.M.
Big Bend, W. Va.
CALDWELL, DAVID E., D.V.M.
102 Western Ave., Findlay, Ohio.
CLOSSON, JOSEPH T., D.V.M.
214 22nd St., Logansport, Ind.
COMPTON, WARREN R., D.V.M.
368 Colonial Lane, Dayton, Ohio.
CONNAIR, THOMAS L., D.V.M.
610 Hodapp Ave., Dayton, Ohio.
DeLONG, EDGAR, C., D.V.M.
3049 Cleveland Ave., Columbus, Ohio.
DROESE, JAMES R., D.V.M.
Antioch Rd., Perry, Ohio.
ENDO, ARTHUR K., D.V.M.
2217 W. 65th St., Cleveland, Ohio.
EPPERSON, M. B., D.V.M.
Galion, Ohio.
EPPLEY, JOHN R., D.V.M.
703 Ridge Ave., Bucyrus, Ohio.
FULLER, RONALD R., D.V.M.
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Rt. 1, Republic, Ohio.
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7015 N. 27th Ave., Phoenix, Ariz.
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104 S. Jefferson St., Pataskala, Ohio.
GRAFF, ALVERDA D., D.V.M.
10007 N. State Rd., Westerville, Ohio.
GRIFFITH, BOB G., D.V.M.
223 E. Jackson St., Georgetown, Ky.
GRUESER, ROGER J., D.V.M.
802 W. Hunter St., Logan, Ohio.
HALE, CHARLES H., D.V.M.
6825 Sodom Hutchings Rd., Girard, Ohio.
HARTMAN, HOWARD A., Jr., D.V.M.
6941 Columbia Rd., Olmsted Falls, Ohio.
HEADLEY, MAURICE G., D.V.M.
Rt. 1, Angola, Ind.
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P. O. Box 73, Harrison, Ohio.
LINERODE, PHILLIP A., D.V.M.
Rochester Animal Hospital, Byron, Minn.
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19110 Springdale Ave., Cleveland, Ohio.
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5500 Glenway Ave., Cincinnati, Ohio.
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1698 Rhoda Ave., Columbus, Ohio.
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School of Veterinary Medicine, Tuskegee Institute, Tuskegee Institute, Ala.
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COMING MEETINGS

American Veterinary Medical Association. Ninety-Fifth Annual Meeting. Convention Hall, Philadelphia, Pa., Aug. 18-21, 1958. J. G. Hardenbergh, 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

Louisiana Veterinary Medical Association, Inc. Annual meeting, Montelone Hotel, New Orleans, Aug. 24-26, 1958. R. R. Gibbs, 6421 Chef Menteur Highway, New Orleans, La., program chairman.

New York State Veterinary Medical Society. Sixty-seventh annual meeting. Concord Hotel, Kiamesha Lake, N. Y., Sept. 4-6, 1958. Miss Joan S. Halat, 803 Varick St., Utica, N. Y., executive secretary.

Colorado Veterinary Medical Association. Annual Meeting. Antlers Hotel, Colorado Springs, Sept. 5-6, 1958. G. H. Gilbert, 5500 Wadsworth Blvd., Arvada, secretary-treasurer.

New Mexico Veterinary Association. Annual meeting. La Pasoda Hotel, Santa Fe, Sept. 8-9, 1958. E. R. Leslie, 920 N. Main, Carlsbad, N. M., secretary.

Washington State Veterinary Medical Association. Annual meeting. Hotel Olympian, Olympia, Sept. 8-10, 1958. F. M. Shigley, P.O. Box 1163, Olympia, Wash., program chairman.

South Dakota Veterinary Association. Meeting. Hotel Cateract, Sioux Falls, Sept. 16-17, 1958. G. E. Duncan, Tyndall, secretary.

New England Veterinary Medical Association. Annual meeting. Hotel Wentworth-By-The-Sea, Portsmouth, N. H., Sept. 28-Oct. 1, 1958. C. Lawrence Blakely, 180 Longwood Ave., Boston 15, Mass., secretary-treasurer.

Missouri, University of. Annual short course for graduate veterinarians. School of Veterinary Medicine, Columbia, Mo., Oct. 6-7, 1958. Cecil Elder, chairman.

Florida State Veterinary Medical Association. Annual meeting. Galt Ocean Mile Hotel, Fort Lauderdale, Oct. 12-14, 1958. A. R. Chambers, 6116 Main St., Jacksonville, secretary.

District of Columbia Veterinary Medical Association. Annual all-day meeting. Walter Reed Army Medical Center, Washington, D.C., Oct. 14, 1958. W. I. Gay, 5200 Chandler St., Bethesda, Md., secretary-treasurer.

Eastern Iowa Veterinary Association, Inc. Annual meeting. Hotel Roosevelt, Cedar Rapids, Oct. 16-17, 1958. F. E. Brutsman, Traer, Iowa, secretary-treasurer.

Texas Veterinary Medical Association. Annual meeting. Stephen F. Austin Hotel, Austin, Oct. 19-21, 1958. Paul B. Blunt, 710 Maverick Bldg., San Antonio, secretary.

Southern Veterinary Medical Association. Annual meeting. Claridge Hotel, Memphis, Tenn., Oct. 26-30, 1958. A. A. Husman, P.O. Box 91, Raleigh, N. Car., secretary.

Arizona Veterinary Medical Association. Annual meeting. Yuma Country Club, Yuma, Dec. 7-9, 1958. Thomas Lightle, Route 1, Box 917, Yuma, Ariz., in charge of reservations.

Tennessee Veterinary Medical Association. Annual meeting.

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Noel Hotel, Nashville, Jan. 11-13, 1959. H. W. Hayes, 5009 Clinton Pike, Knoxville, secretary-treasurer.

Oklahoma Veterinary Medical Association. Annual meeting. Mayo Hotel, Tulsa, Jan. 25-27, 1959. M. N. Riemenschneider, 122 State Capitol Bldg., Oklahoma City, secretary.

Ohio State Veterinary Medical Association. Annual convention. Neil House Hotel, Columbus, Feb. 4-6, 1959. Harry C. Sharp, 1411 W. Third Ave., Columbus, Ohio, executive secretary.

Foreign Meetings

Sixth International Congresses on Tropical Medicine and Malaria. Lisbon, Portugal, Sept. 5-13, 1958. Professor Manuel R. Pinto, Institute of Tropical Medicine, Lisbon, secretary-general. (Membership application forms may be obtained by U.S. veterinarians by writing to the AVMA.)

Veterinary Congress of Brussels, Brussels, Belgium, Sept. 6-17, 1958. Further information available from Bureau, 35, Rue de la Brasserie, Brussels 5.

German Veterinary Association. Regular biennial meeting. Hannover, Germany, Sept. 19-21, 1958. Dr. Karl Ohly, 123 Forsthausstrasse, Frankfurt/Main, Germany, president.

Seventh International Congress on Hydatid Disease, Beirut, Lebanon, Sept. 20-22, 1958. Dr. Elias Sader, c/o Lebanese Order of Physicians, P.O.B. 640, Beirut, Lebanon, secretary-general. (Applications to present papers must be accompanied by an abstract).

International Veterinary Congress. Sixteenth session. Madrid, Spain, May 21-27, 1959. Prof. Pedro Carda A., general secretary, Calle Villanueva 11, Madrid.

U.S. COMMITTEE: Dr. W. A. Hagan, chairman, New York State Veterinary College, Ithaca, N. Y.; Dr. J. G. Hardenbergh, secretary, 600 S. Michigan Ave., Chicago 5, Ill.

Third World Congress on Fertility and Sterility. Amsterdam, Holland, June 7-13, 1959. Dr. L. I. Swaab, Sint Agnietenstraat 4, Amsterdam, Holland, honorary secretary.

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. Dr. G. W. Jones, Main St., Prattville, Ala., secretary-treasurer.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

North Alabama Veterinary Medical Association, the second Thursday of November, January, March, May, July, and September, in Decatur, Ala. Ray A. Ashwander, Decatur, Ala., secretary.

North East Alabama Veterinary Medical Association, the second Tuesday of every other month. Leonard J. Hill, P.O. Box 761, Gadsden, Ala., secretary-treasurer.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2 Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

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Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December. Herb Warren, 3004 16 St., San Francisco, Calif., executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. James L. Frederickson, 17 Niles St., Bakersfield, Calif., secretary-treasurer.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643-N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. R. M. Granfield, 2600 W. El Camino Real, San Mateo, Calif., secretary-treasurer.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert L. Chandler, P.O. Box 8, Ukiah, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Chapter SCVMA, the second Tuesday of each month at 7:30 p.m., Hody's Restaurant, North Hollywood, Calif. Dr. V. H. Austin, 14931 Oxnard St., Van Nuys, secretary-treasurer.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. Dr. Rolf Reese, 23815 Ventura Blvd., Calabasas, Calif., secretary.

Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Beulley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinary Medical Association, the second Thursday of each month. D. E. Britten, 544 N. Ben Maddox, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of

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Pfizer

Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Central Florida Veterinary Medical Association, the first Tuesday of each month, time and place specified monthly. Jack H. McElyer, 5925 Edgewater Drive, Orlando, Fla., secretary.

Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, St. Petersburg. William F. Casler, 2540 30th Ave., N., St. Petersburg, secretary-treasurer.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodsons Restaurant. P. S. Roy, 4443 Atlantic Blvd., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. T. R. Geci, 1088 Catherine Ave., Pensacola, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. J. J. McCarthy, 500-25th Street, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Frank Mueller, Jr., 4148 E. 8th Ave., Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. W. B.

Dr. R. N. Nelson to Bio Laboratories

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Dr. Ralph N. Nelson

graduate of microbiology at the University of Toronto in 1949, he has worked for the past four years as biological production manager for Diamond Laboratories in Des Moines, Iowa.

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Martin, Jr., 3002 N. W. 6th St., Gainesville, Fla. secretary.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. A. E. Hixon, 131 Mary St., Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the third Thursday of each month at the Elk's Home, 726 Peachtree St., Atlanta. Donald C. Ford, Forest Park, secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Charles H. Armstrong, 1021 Davis St., Evanston, secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. Alfred G. Schiller, Veterinary Clinic, University of Illinois, Urbana, secretary-treasurer.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 342½ S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P.O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Medical Association, the second Monday of each month, except January, July, August, and October in Black's Tea Room, Waterloo, Iowa. A. J. Cotten, Grundy Center, secretary.

Central Iowa Veterinary Medical Association, the third Monday of each month, except June, July, and August, at 6:30 p.m., Breeze House, Ankeny, Iowa. John Herrick, Ames, secretary.

Coon Valley Veterinary Medical Association, the second Wednesday of each month, September through May, at 7:30 p.m., Cobblestone Inn, Storm Lake, Iowa. Robert McCutcheon, Holstein, secretary.

East Central Iowa Association, the second Thursday of each month at 6:30 p.m., usually in Cedar Rapids, Iowa. Dr. J. G. Irwin, Iowa City, secretary.

Fayette County Veterinary Medical Association, the third Thursday of each month at 6:30 p.m. in West Union, Iowa. H. J. Morgan, West Union, secretary.

Lakes Veterinary Association, the first Tuesday of each month, September through May, at 6:30 p.m., at the Gardson Hotel, Estherville, Iowa. Barry Barnes, Milford, secretary.

North Central Iowa Veterinary Medical Association, the third Thursday of April, at the Warden Hotel, Fort

(Continued on adv. p. 42)

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1. Belloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956. 2. Mosier, J. E.: Vet. Med. 52:445 (Sept.) 1957.

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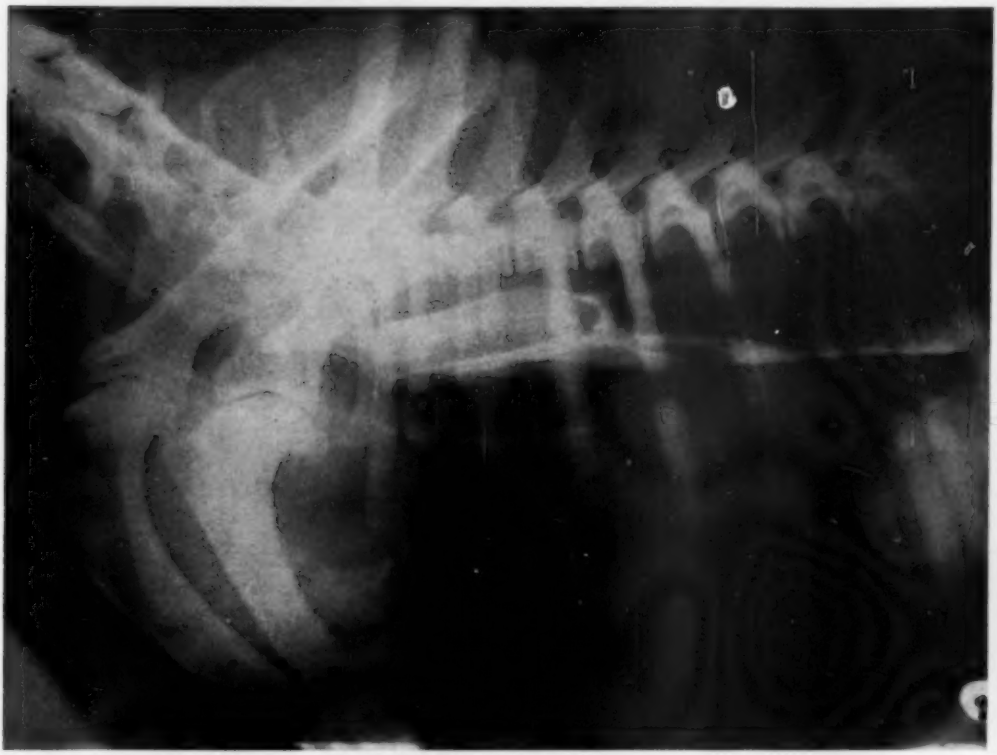

Figure 1

History.—A male Beagle, 2 years old, and in good health, was turned out-of-doors unattended. Upon his return, he did not eat and later, when he did, the food was regurgitated almost immediately. Radiographs of the dog were taken. Shown here is the photograph of one taken from a lateral view within a few minutes after a thin barium solution (0.5 ounce) was given by mouth.

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—A foreign body present in the esophagus in the region constricted by the aortic arch and accompanying structures at the base of the heart.

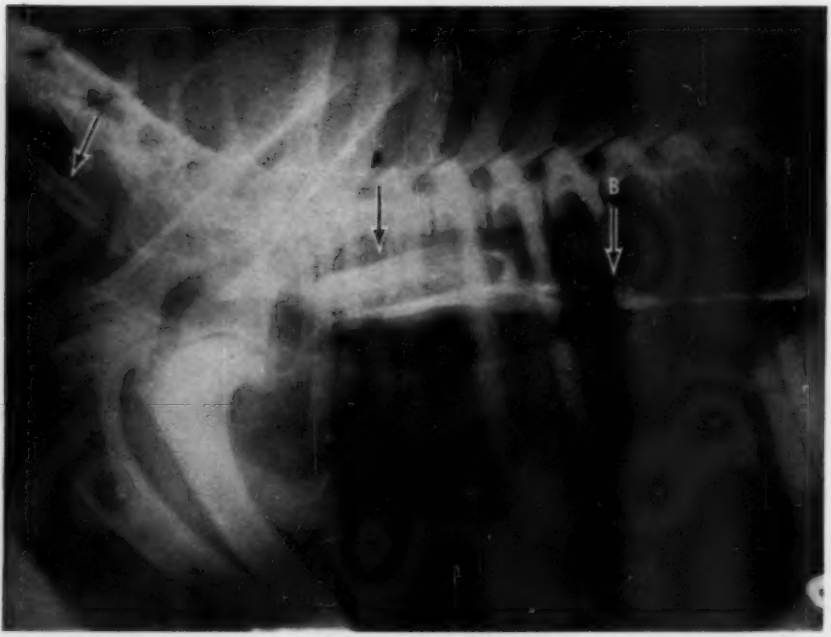

Fig. 2—Photograph of a radiograph, lateral view, of the thorax showing the piece of bone (a) in the esophagus at the constriction of the aortic arch anterior to the base of the heart; barium solution in the esophagus (b).

Editor's Comment.—The foreign body, which was removed by forceps by way of the mouth, proved to be a piece of bone. In the dog, the esophagus is somewhat constricted just anterior to the base of the heart and again immediately in front of the diaphragm. Obstruction usually occurs at one of these two regions.

This case was submitted by Dr. Burton Saunders, Yorktown Heights, N. Y.

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Dodge, Iowa. H. Engelbrecht, P. O. Box 797, Fort Dodge, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northwest Iowa Veterinary Medical Association, the second Tuesday of February, May, September, and December, at the Community Bldg., Sheldon. W. Ver Meer, Hull, secretary.

Southeastern Iowa Veterinary Association, the first Tuesday of each month at Mt. Pleasant, Iowa. Warren Kilpatrick, Mediapolis, secretary.

Southwestern Iowa Veterinary Medical Association, the first Tuesday of April and October, Hotel Chieftain, Council Bluffs, Iowa. J. P. Stream, Creston, secretary.

Upper Iowa Veterinary Medical Association, the third Tuesday of each month at 7:00 p.m., at All Vets Center, Clear Lake, Iowa. Richard Baum, Osage, secretary.

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirrell, Varsailles Rd., Frankford, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday of each month in Louisville or within a radius of 50 miles, except January, May, and July. G. R. Comfort, 2102 Reynolds Lane, Louisville, Ky., secretary-treasurer.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m., at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Norman Herbert, 3506 Joann Drive, Baltimore 7, Md., secretary.

MICHIGAN—Central Michigan Veterinary Medical Association, the first Wednesday of every month at 7 p.m. Frank A. Carter, P.O. Box 78, Carson City, Mich., secretary.

Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Edwin E. Epstein, 4877 Natural Bridge Ave., St. Louis 15, Mo., secretary.

Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Thursday of each month at the Hotel President, Kansas City, Mo. Frank A. O'Donnell, Parkville, Mo., secretary-treasurer.

NEVADA—Western Nevada Veterinary Society, the first Tuesday of each month. Paul S. Silva, 1170 Airport Road, Reno, Nev., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Elks Club, Hackensack. Burritt Lupton, 569 Franklin Ave., Wyckoff, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. R. Muller, 43 Church St., Lambertville, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the fourth Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. R. M. Sauer, secretary.

NEW MEXICO—Bernalillo County Veterinary Practitioners Association, third Wednesday of each month, Fez Club, Albuquerque, N.M. Jack Ambrose, 3018 N. Rio Grande Blvd., Albuquerque, secretary-treasurer.

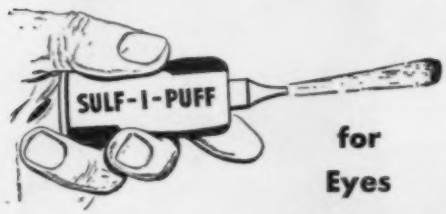
NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

New York State Veterinary College, Annual conference for veterinarians. Cornell University, Ithaca. W. A. Hagan, New York State Veterinary College, Cornell University, Ithaca, N. Y., dean.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month, time and place specified monthly, Byron H. Brow, Box 453, Goldsboro, N. Car., secretary.



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Piedmont Veterinary Medical Association, the last Friday of each month. T. L. James, Box 243, Newton, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Friday of each month at Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. J. E. Currie, 690 N. Leak St., Southern Pines, N. Car., secretary.

Western North Carolina Veterinary Medical Association, the second Thursday of every month at 7:00 p.m. in the George Vanderbilt Hotel, Ashville, N. Car. Vilu Lind, 346 State St., Marion, N. Car., secretary.

OHIO—Cincinnati Veterinary Medical Association, the third Tuesday of every month at Shuller's Wigwam, 6210 Hamilton Ave., at North Bend Road. G. C. Lewis, Cincinnati, Ohio, secretary-treasurer.

Columbus Academy of Veterinary Medicine, every month, September through May. E. M. Simonson, Columbus, Ohio, secretary-treasurer.

Cuyahoga County Veterinary Medical Association, the first Wednesday in September, October, December, February, March, April and May, at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. F. A. Coy, Cleveland, Ohio, secretary.

Dayton Veterinary Medical Association, the third Tuesday of every month. O. W. Fallang, Dayton, secretary.

Killbuck Valley Veterinary Medical Association, the first Wednesday of alternate months beginning with February. D. J. Kern, Killbuck, Ohio, secretary-treasurer.

Mahoning County Veterinary Medical Association, the third Tuesday of each month, at 9:00 p.m., Youngstown Maennerchor Club, Youngstown, Ohio. Sam Segall, 2935 Glenwood Ave., Youngstown, secretary.

Miami Valley Veterinary Medical Association, the first Wednesday of December, March, June, and September. J. M. Westfall, Greenville, Ohio, secretary-treasurer.

North Central Ohio Veterinary Medical Association, the last Wednesday of each month except during the summer. R. W. McClung, Tiffin, Ohio, secretary-treasurer.

Northwestern Ohio Veterinary Medical Association, the last Wednesday of March and July. C. S. Alvanos, Toledo, Ohio, secretary-treasurer.

Stark County Veterinary Medical Association, the second Tuesday of every month, at McBrides Emerald Lounge, Canton, Ohio. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

Summit County Veterinary Medical Association, the last Tuesday of every month (except June, July, and August), at the Mayflower Hotel, Akron, Ohio. M. L. Scott, Akron, Ohio, secretary-treasurer.

Tri-County Veterinary Medical Association, the fourth Wednesday of January, May, and September. Mrs. R. Slusher, Mason, Ohio, secretary-treasurer.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Forest H. Stockton, 2716 S.W. 29th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

OREGON—Portland Veterinary Medical Association, the second Tuesday of each month, at 7:30 p.m., Ireland's Restaurant, Lloyds', 718 N.E. 12th Ave., Portland. Donald L. Moyer, 8415 S.E. McLoughlin Blvd., Portland 2, Ore., secretary.

Willamette Veterinary Medical Association, the third Tuesday of each month, except July and August, at the Marion Hotel, Salem. Marvin M. Corff, McMinnville, Ore., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania, School of Veterinary Medicine. Raymond C. Snyder, N.E. Corner 47th St. and Hazel Ave., Philadelphia 43, Pa., secretary.

Lehigh Valley Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

Pennsylvania Northern Tier Veterinary Medical Association, the third Wednesday of each odd numbered month. R. L. Michel, Troy, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary Ct., Richmond 20, Va., secretary.

Northern Virginia Veterinary Conference, the second Tuesday of each month. Francis E. Mullen, 1130 S. Main St., Harrisonburg, Va., secretary-treasurer.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 143, Merrifield, secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blackburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle, Wash. William S. Green, 9637 S. E. 36th, Mercer Island, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. O. I. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Pritchard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

Prehistoric Dog Found in Germany

The well-preserved body of a dog which died about 3,200 years ago was recently found in northwestern Germany. It was of the Spitz type, slightly smaller than the modern Norwegian Elkhound, with black-brown fur 1 to 1½ inches long and a light woolly undercoat, pricked ears, and a curly tail.

A few years ago, in the peat lands of Denmark, another such dog was found, so well preserved that it seemed to be asleep. It wore a fiber collar and was thought to be a hunting dog of a tribe that inhabited the land just after the ice age. Since weapons of that time were inferior (the bow and arrow had not yet been invented), the hunting dog was important to the survival of these northern tribes and a good dog was highly prized. When they died, they were buried either in peat, where rodents and worms can not live, or the body was protected from predators by a pyramid of stones built over the grave.—*Dogs in Canada (May, 1958): 10.*

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Tubes of 3.5 Gm. Each gram of ointment contains: 2.5 mg. prednisolone 21-phosphate 5 mg. neomycin sulfate.	**Ointment** Apply a small quantity inside the lower lid three times a day.
Tubes of 5 Gm. Each Gm. of ointment contains: 25 mg. of hydrocortisone 5 mg. neomycin sulfate 1,000 units of zinc bacitracin	**Topically:** Two or three applications daily

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WISCONSIN—Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.). D. F. Ludvigson, Ridgeland, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Farley Ave., Madison, Wis., secretary.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. R. H. Steinkraus, 7701 N. 59th St., Milwaukee, Wis., secretary.

Northeastern Wisconsin Veterinary Medical Association, the third Wednesday in April. William Madson, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. W. E. Lyle, P. O. Box 107, Deerfield, Wis., secretary.

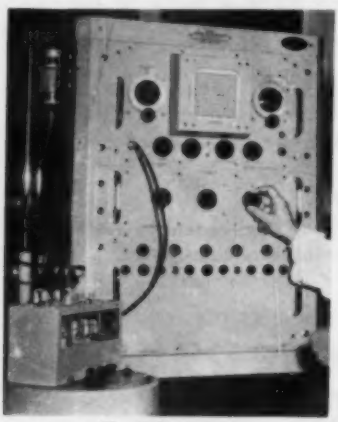
Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

Wisconsin Valley Veterinary Medical Association, the second Tuesday of every other month. E. S. Scobell, Rt. 2, Wausau, Wis., secretary.

Unusual Application of Polarography

A unique use of an instrument, the Polarotrace, is made at the Institute of Medical and Veterinary Science, Adelaide, Australia.

Dr. P. S. Watts, head of the Institute's Veterinary Division, states that the maximum production of wool and meat from sheep depends largely on adequate trace mineral intake. Trace metals



The Polarotrace

amounting to one-hundredth of a millionth part in animal body fluid can be determined by the Polarotrace.

The trace elements are cobalt, copper, manganese, and zinc. Methods are being developed for iron and phosphorus. The instrument is manufactured by Southern Instruments, England, and distributed by Standard Scientific Supply Corporation in the United States.

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Names of classified advertisers using key letters can not be supplied. Address your reply to the box number, c/o JOURNAL of the AVMA, 600 S. Michigan Ave., Chicago 5, Ill., and it will be sent to the advertiser.

Wanted—Veterinarians

Veterinarian wanted—to assist in mixed practice (predominantly small animal) in modern, completely-equipped hospital in southern city. Please state experience. Salary open. Address "Box M 25," c/o JOURNAL of the AVMA.

Wanted—personable assistant for a modern small animal hospital in the Southwest. State age, qualifications, and references in letter. Address "Box M 32," c/o JOURNAL of the AVMA.

Wanted—assistant veterinarian for staff of humane organization, New York. State license required, recent graduate preferred. Excellent experience, salary, and opportunity for advancement for right man. Address "Box M 37," c/o JOURNAL of the AVMA.

Wanted—career state civil service field veterinarians, $6,444-$8,424, vacation, sick leave, retirement benefits. Ages 21-50, graduate accredited college; U.S. citizenship required. Contact Animal Health Division, State Capitol, Madison, Wis.

Veterinarian, licensed in Maryland, to assist small animal practice, Baltimore. Recent graduates acceptable. Growing practice; partnership possibility. State qualifications. Address "Box M 41," c/o JOURNAL of the AVMA.

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Graduate (Minn. '54), married, military service being completed, desires position in mixed or large animal practice. Prefer practice utilizing airplane. Address "Box M 44," c/o JOURNAL of the AVMA.

Experienced Texas graduate desires position in established mixed practice. Address "Box M 26," c/o JOURNAL of the AVMA.

D.V.M. (1955), experienced, desires association in general practice. Married; military obligation completed September. Prefer Middlewest or New England areas. Address "Box M 35," c/o JOURNAL of the AVMA.

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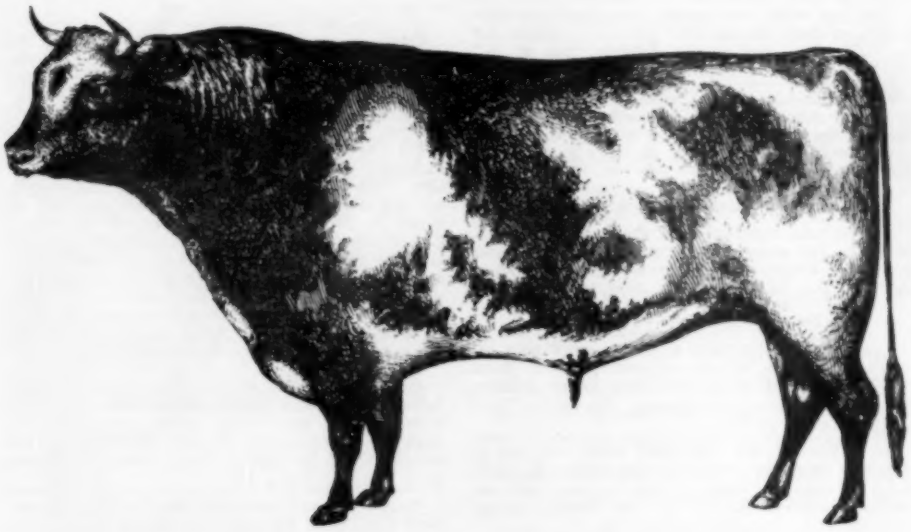

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Cornell graduate (1955), desires position in mixed or small animal practice. Licensed in New York, New Jersey, and Connecticut. Military obligations completed. Address "Box M 40," c/o JOURNAL of the AVMA.

Wanted—Practices

Veterinarian, experienced in small animal practice, wants to buy active animal hospital. Would prefer central or western states, but consider any location. Address "Box M 39," c/o JOURNAL of the AVMA.

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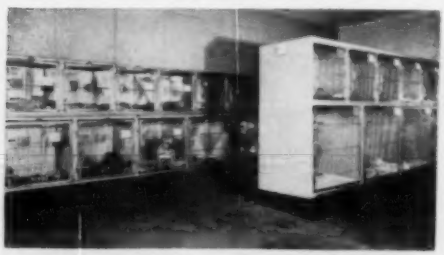
New hospital and 3-bedroom home for sale in southern Michigan. Thriving mixed practice; photo sent on request. Owner leaving for other interest. $40,000. Address "Box M 28," c/o JOURNAL of the AVMA.



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For sale—mixed practice, east central Illinois. Sales includes real estate, medical coach on 1958 GMC chassis, Silver King cattle chute, and other large animal equipment. $13,250; $6,750 down will handle. Write Dr. Guy C. Poppy, Box 216, Paxton, Ill.

Prosperous, well-established small animal hospital, New York State. Includes valuable real estate, 4-bedroom home. Sacrificing for quick sale. Address "Box M 33," c/o JOURNAL of the AVMA.

Virginia mixed practice—in prosperous community. Price includes hospital, fully-equipped, and apartment. Priced at value of physical assets. Reason for selling: health. Address "Box M 34," c/o JOURNAL of the AVMA.

Miscellaneous

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Assistantship desired with small animal practitioner in New Jersey or Connecticut. Licensed both states. Address "Box M 42," c/o JOURNAL of the AVMA.

Wanted—veterinarian with Oregon license to assist in mixed practice in southwest Oregon. Some insemination; prefer recent graduate. Car furnished, good starting salary to right party. Address "Box M 43," c/o JOURNAL of the AVMA.

For sale—good mixed practice in east central Michigan. Large and small animal hospital, 2-bedroom home. Ill health reason for selling. Address "Box M 45," c/o JOURNAL of the AVMA.

26-year-old established practice for sale in south central Minnesota. 5-bedroom brick home and 2-car block garage; office in home. Price: $24,000; one-half down. Address Dr. E. H. Enge, Comfrey, Minn.

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Index to Advertisers in This Issue

Abbott Laboratories 49
Affiliated Laboratories Corporation ... 17
Armour Veterinary Laboratories 29
Associated Veterinary Laboratories . 18, 19
Bandy Laboratories, Inc. 37
Birtcher Corp., The 7
Ciba Pharmaceutical Products, Inc. 8, 9, 47
Colwell Publishing Co. 4
Corn Belt Laboratories, Inc. 36
Corn States Laboratories, Inc. .. 2nd cover
Curtis Laboratories 42
Eaton Laboratories 12, 13, 38
Fort Dodge Laboratories 22
G.L.F. 32
Hill Packing Company 11
Hyland Laboratories 25
Jensen-Salsbery Laboratories, Inc. .....
..... 41, 4th cover
Liability Insurance 35
MacAllan Laboratories 24
Massengill Company, S. E. 6
Merck & Co., Inc. 44, 45
National Laboratories Corporation 10
Norden Laboratories 1
Ohio Chemical 36
Parke, Davis & Company 23
Pet Chemicals, Inc. 33
Pfizer Laboratories 15, 34
Pitman-Moore Company ..... 3, 3rd cover
Professional Printing Company, Inc. .. 14
Research Laboratories, Inc. 32
Sani-Cage Distributing Co. 48
Swift and Co. 21
Upjohn and Company 27
Warner-Chilcott Laboratories 20
Westwood Pharmaceuticals 5
Wyeth 50

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Exclusive Publication.—Articles submitted for publication are accepted with the understanding that they are not submitted to other journals, which is ethical publication procedure.

Manuscripts.—Manuscripts, including **footnotes, references,** and **tables,** must be typewritten, **double-spaced,** on 8½- by 11-in. bond paper, and the original and one carbon copy, submitted. One-inch margins should be allowed on the sides, with 2 in. at top and bottom. Articles should be concise. Short, simple sentences are clearer and more forceful than long, complex ones.

Illustrations.—Photographs should be furnished in glossy prints, and of a size that will fit into the JOURNAL of the American Veterinary Medical Association with a minimum of reduction. Photomicrographs which can not be reduced should be marked for cropping to 1-column or 2-column width. Identifying marks within the photomicrographs, such as arrows, letters, or numbers, should be clearly marked with black India ink or white opaque ink to insure good contrast for reproduction and must be large enough to stand reduction, if necessary.

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Tables.—Tables should be simple and typed **double space.** Complex tables are not conducive to perusal. It is wiser to summarize complex material than to tabulate it.

References.—References should be typed **double space,** in alphabetical order, and should be prepared in the following style: name of author, title of article, name of periodical with volume, year, and page numbers. References to journals not commonly known should give the complete name of the periodical, and where published so that they may be added to our reference files. When books are cited, the name of publisher, location, edition, and year should be given.

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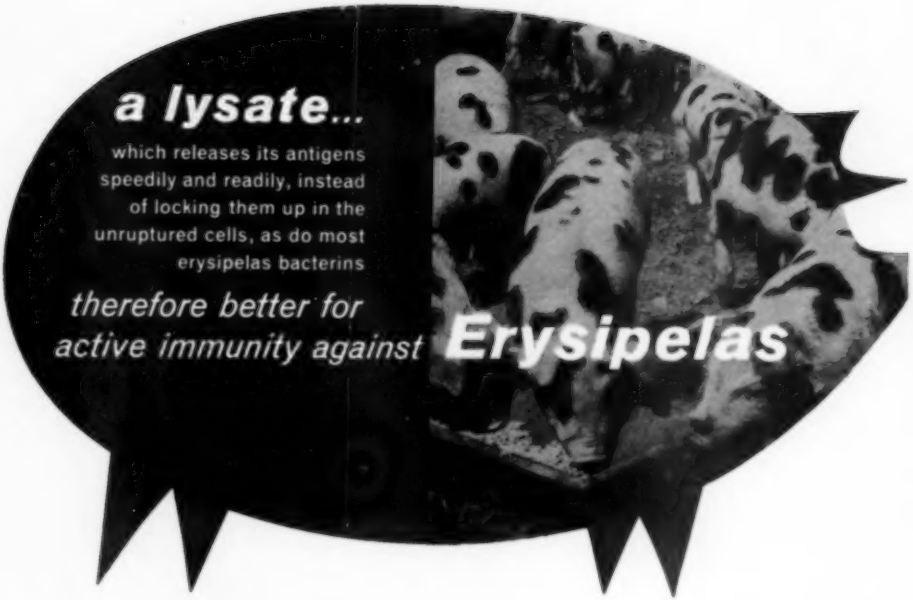
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